VOLUME 70 • NOVEMBER 1959 • NUMBER 5

A.M.A. ARCHIVES OF

OTOLARYNGOLOGY

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Plastic Surgery
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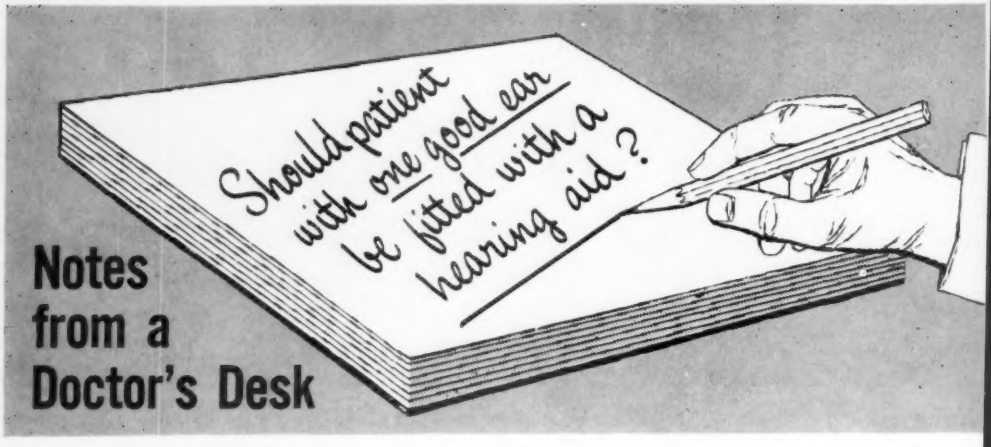

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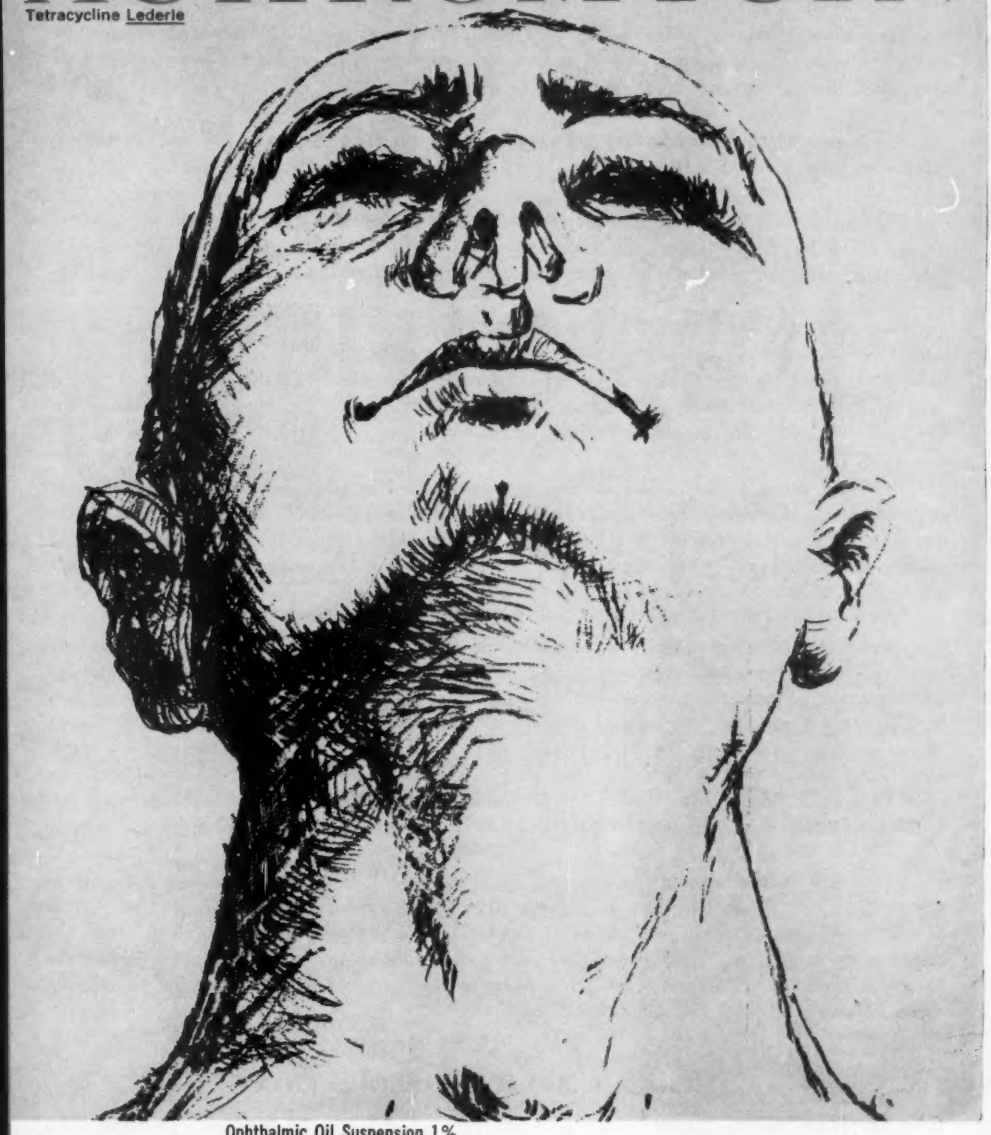

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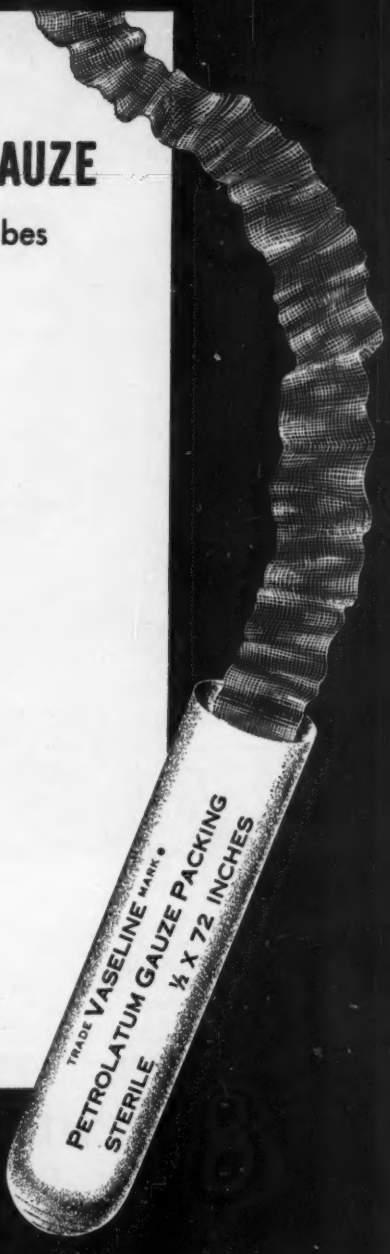

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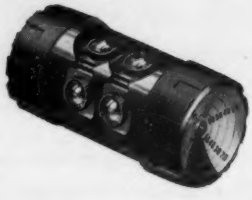

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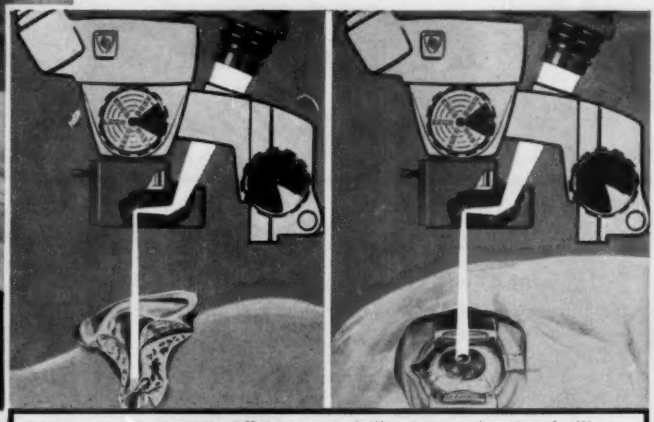

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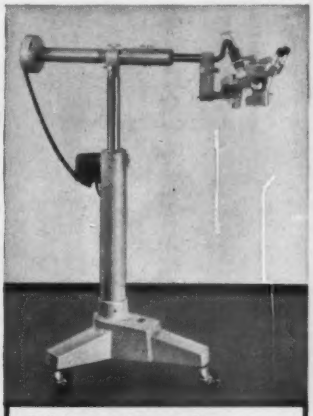

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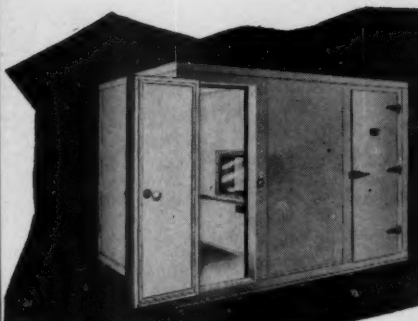

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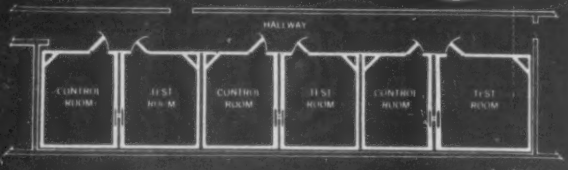

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1. Jones, E. H.: Eye, Ear, Nose and Throat Monthly *38*:460, 1959.

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The Relation of Endemic Goiter to Deaf-Mutism

Isidor Greenwald, Ph.D., New York

In another paper, I [1] called attention to the large number of deaf-mutes found in those parts of Peru and of the Argentine Republic in which goiter was most prevalent. In such districts, the number of deaf-mutes greatly exceeded the number of cretins and idiots, and they did this to such a degree that the excess could not have been due to other known causes of deaf-mutism but was probably related to goiter.

The relation between endemic goiter and deaf-mutism was once well recognized. Baillarger [2] gave it considerable attention, and H. Bircher [3] devoted one-third of his book to it. Since then, the subject has almost disappeared from the literature concerning goiter. Most recent books on the thyroid and its diseases either fail to mention deaf-mutism or refer to it only as one of the manifestations of cretinism.

Writers on deaf-mutism have taken much the same attitude. Thus, Watkyn-Thomas [4] wrote: "The endemic cretinous cases (Siebenmann's type) are usually feeble-minded and often goitrous from birth. Goiter is often present in other members of the family. Occasionally, but rarely, the deafness is the only symptom of cretinoid degeneration." Designating deaf-mutism in a person of otherwise apparently normal intelligence as "the only symptom of cretinoid degeneration" is begging the question of whether or not deaf-mutism and cretinism are independent manifestations or accompaniments of dysfunction of the thyroid.

More recently, the association of deaf-mutism with familial goiter has attacted some attention (Brain,[5] Deraemaeker,[6] Morgans and Trotter,[7] Thieme [8]). Indeed there have been several reports of difficulties in hearing that developed in adult life together with myxedema or with goiter, or both, and that were benefited by treatment with thyroid. (Howarth and Lloyd,[9] Kemp,[10] King,[11] Moehlig,[12] Rau,[13])

In the present paper, I have brought together such pertinent data for the prevalence of goiter, deaf-mutism, and cretinism as I could find. They indicate very clearly that deaf-mutism, in regions in which goiter is common, is *not* dependent upon cretinism.

So far as I have been able to determine, no such high proportion of deaf-mutes, more than 5 per 1,000 (in one department almost 20), as was reported from four departments of the Argentine Republic in 1869 (Table 1) and from four departments of Peru in 1940 (Table 2) have ever been reported from other territories of similar size or population and from but few of much smaller area and number of inhabitants.

It will be noted (Table 1) that, in the one department of the Argentine Republic in which the proportion of goiters increased from 1869 to 1895, there was also a slight increase in the number of deaf-mutes, although the number of idiots had diminished.

In the paper to which reference has already been made,[1] it was shown that the

Submitted for publication April 28, 1959.
New York University College of Medicine.
Aided by grants from the National Institutes of Health and the National Science Foundation.

TABLE 1.—*Data from Eight Departments of the Argentine Republic*[14] *

Department	Goiters		Idiots		Deaf-Mutes		
	1869	1895	1869	1895	1869	1895	1914
Mendoza	2,712	1,829	508	191	1,321	654	252
Jujuy	1,325	1,147	854	288	1,946	1,160	673
Salta	1,127	538	912	239	1,287	709	700
Tucuman	552	211	309	90	491	217	166
Catamarca	279	872	309	120	354	372	176
Capital	20	11	64	37	118	54	67
Buenos Aires	25	21	139	43	169	56	61
Santa Fe	20	21	90	27	170	46	47

* No. persons with obvious goiters and number of idiots including cretins per 100,000 of population in 1869 and 1895 (2d census); no. deaf-mutes per 100,000 in 1869, 1895, and 1914 (3d census). (By 1914, the number of goiters had diminished so greatly that no separate enumeration was believed necessary.).

association of a deaf-mutism with goiter had been observed, or surmised, in the 1820's, although the presence of cretinism was explicitly denied.

In 1864, in the most goitrous department of France (Savoie), with goiter apparently four times as frequent as in the most severely affected departments of Peru or of the Argentine Republic (compare Table 3 with Tables 1 and 2) the proportion of deaf-mutes to the total population was only one-third as great. In Haute-Savoie, with 9.2% of goiters, the proportion of deaf-mutes was of the same order as in almost goiter-free regions. In the three most goitrous departments, cretins and idiots outnumbered deaf-mutes by from 2.9 more than 10:1. This was very different from the situation in South America.

There are no really comparable data from either Italy or Switzerland. However, the Swiss census of 1870 did include figures for the number of deaf-mutes and of *Geisteskranken* (those with mental disease), which, presumably, included cretins. I have calculated the proportions of both in the various cantons and have arranged the data in order of decreasing proportion of deaf-mutes (Table 4). Here we again have a considerably greater number of deaf-mutes than of "Geisteskranken" in the five cantons with the greatest proportion of deaf-mutes.

There are also included in Table 4, figures for the proportion of young men called for military service in 1884, 1885, and 1886 that were rejected because of goiter. These data are for a period from 14 to 16 years after those preceding them in the Table and a perfect correspondence is not to be expected. However, if we divide the 22 cantons into groups having in 1870, (1) more than four deaf-mutes per 1,000, (2) those with from 1.5 to 2.4, (3) those with from 1.24 to 1.48 and (4) those with 1.15 or less, we obtain the figures in the last column in the Table. The correlation between rejections for goiter and the

TABLE 2.—*Data from Seven Departments of Peru*[15] *

Department	Goiters	"Dementes"	Deaf-Mutes
Amazonas	2,879	200	843
Huanuco	1,996	134	669
Ancash	1,436	168	649
Junin	509	112	397
Lambayeque	5	77	86
Ica	7	68	72
Tacna	8	94	105

* No. persons with obvious goiters, of "dementes," and of deaf-mutes per 100,000 of population in 1940.

TABLE 3.—*Data from Seven Departments of France* *

Department	Goiters	Idiots & Cretins	Deaf-Mutes
Savoie	13.37	1.60	0.264
Hautes-Alpes	11.10	2.25	0.222
Haute-Savoie	9.20	0.45	0.165
Gironde	0.24	0.19	0.152
Cher	0.20	0.28	0.164
Pas de Calais	0.16	0.26	0.170
Indre et Loire	0.08	0.26	0.183

* No. persons with obvious goiters and number of idiots, cretins, and deaf-mutes per 100 of population in 1864 (Baillarger [9]).

TABLE 4.—*Data from Twenty-Two Cantons of Switzerland* *

Canton	Deaf-Mutes	"Geistes-kranken"	Rejected Because of Goiter	
Wallis (Valais)	4.02	2.02	94	
Luzern	4.36	2.38	213	120
Aargau	4.35	3.02	139	
Bern	4.16	3.99	108	
Freiburg	4.03	3.39	246	
Graubunden	2.42	2.16	69	
Uri	2.33	1.61	74	98
Appenzell	1.96	2.05	198	
Glarus	1.53	1.48	46	
St. Gallen	1.51	2.54	90	
Basel	1.48	2.00	83	
Zurich	1.31	3.97	113	
Unterwalden	1.28	2.52	79	77
Waadt	1.28	2.46	27	
Solothurn	1.26	3.57	78	
Schaffhausen	1.24	2.04	83	
Tessin	1.15	1.96	20	
Zug	1.13	1.09	172	
Thurgau	0.93	3.47	64	54
Schwyz	0.90	1.80	132	
Neuenburg	0.41	1.56	32	
Geneva	0.27	1.61	25	

* Comparison of proportion of deaf-mutes in total population with "Geisteskranken" (those with mental disorders) and with proportion of young men rejected for military service because of goiter in 1884, 1885, and 1886, in the Swiss Cantons. All figures are per thousand. [14]

proportion of deaf-mutes is obvious. Even without such grouping, the coefficient of correlation between the figures of deaf-mutism and those for rejections because of goiter is 0.7.

Bircher [3] made a survey of the incidence of goiter among the school-children in the various communities of the district of Aarau, canton of Aargau. I have selected from his data those that refer to communities that had, in 1870, a population of 1,000 or more. (Smaller groups are not likely to give significant statistics.) These data are presented in Table 5, together with those for the proportion of deaf-mutes and "Geisteskranken" in 1870. It is apparent that there are close correlations between the figures for goiters, for deaf-mutism, and for cretinism. It is also clear that, in goitrous communities, the number of deaf-mutes was greater than that of cretins. In these respects, the situation in that part of Switzerland, at that time, resembled that in Peru in 1940 and in the Argentine Republic in 1869 and 1895.

In Table 6, there are presented figures for the number of deaf-mutes per 100,000 of total population in several states of the United States in 1910, 1920, and 1930 and in several provinces of Canada in 1911, 1921, and 1931. There were no significant differences between the states and provinces in the "goiter belt" and those immediately contiguous to the ocean. The *highest* figures were of the same order as the *lowest* in Peru or in the Argentine Republic and were lower than those for all but few cantons of Switzerland in 1870. It is probable that these low figures for the United States and Canada in recent years were due to the lesser occurrence of deafness (and mutism) due to bacterial infections in early life.

These differences in the relations between goiter and deaf-mutism in different countries are too great to be due to differences in diagnoses. They are real and should

TABLE 5.—*Data from District of Aarau, Canton of Aargau, Switzerland* *

Community	Population	1843			1870	
		Goiters in School Children, %	Deaf-Mutes, %	Cretins, %	Deaf-Mutes, %	Geisteskranken, %
Buchs	1,000	51	4.5	0.7	1.0	1.3
Gränichen	2,732	42	1.5	0.9	1.5	0.5
Suhr	1,425	37	2.8	1.2	2.0	1.3
Ober-Entfelden	1,311	32	1.0	0.4	1.0	0.4
Mühen	1,273	25	2.3	1.6	1.1	0.3
Denspüren	1,075	3			0.2	0.2
Küttingen	1,817	3	0.1	0.1	0.1	0.2

* Proportion of school children with goiter, as reported by Bircher, [3] of deaf-mutes and cretins in total population in 1843 [17] and of deaf-mutes and *Geisteskranken* in total population in those communities of the district of Aarau, in the canton of Aargau, having 1,000, or more, inhabitants in 1870. [18]

TABLE 6.—*Data from United States and Canada**

State	1910	1920	1930
Ohio	56	48	46
Michigan	49	48	49
Wisconsin	56	64	60
Minnesota	54	41	48
Florida	29	41	49
South Carolina	49	49	32
Rhode Island	28	53	30
Canada	**1911**	**1921**	**1931**
Manitoba	65	45	67
Saskatchewan	37	34	39
Alberta	39	28	40
Nova Scotia	96	83	89
Prince Edward Island	50	45	51

* Deaf-mutes per 100,000 of population in 1910, 1920, and 1930 in four states in the "goiter-belt" and in three states immediately contiguous to the ocean [18]; also in 3 prairie and two maritime provinces of Canada. [19]

be taken into account in any consideration of the causes of these conditions.

There appear to be other differences in the accompaniments of endemic goiter in different regions. For instance, human neonatal goiter seems to be much less frequent in the United States than in Europe. On the other hand, widespread goiter in farm-animals, such as that accompanying "hairlessness" in young pigs, has been relatively common in parts of the United States and Canada but has only recently made its appearance in southern Germany, Austria, and Japan, and has never been reported from other parts of the world.

How a lack of iodine should have such different consequences on two sides of the Atlantic, or in North versus South America, is not apparent. The facts here presented add to the evidence that endemic goiter is not due to a lack of iodine but to some other agent, or agents, that need not be the same in all places and at all times.

In this paper, there has been no consideration of the anatomical changes that underlie the loss of hearing that is associated with goiter. Bircher had discussed these. The evidence was, and still is, scanty. It is unfortunate that the abundant material available in South America has not been utilized.

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Bilateral Nasal Proboscis

Associated with Unilateral Anophthalmia, Unilateral Diffuse Pigmentation of the Conjunctiva, and Anomalies of the Skull and Brain

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Cases in which there is a lateral nasal proboscis on one side have been described and illustrated fairly frequently. The early literature has been reviewed by Grunberg (1909), Zausch (1926), Berblinger (1928), and Stupka (1938). Cases have been described also by Blair and Brown (1931), Biber (1949), Smith (1950), Cottle et al. (1956), Meyer (1956), and Bremer (1956). The case presented here had a lateral nasal proboscis on each side; we have found no reference to such an instance and thus consider it worth while reporting. We have called the condition bilateral nasal proboscis.

Report of a Case

The mother of the infant to be described was 39 years of age. Her previous two children are alive and well with no abnormalities. During her third pregnancy, which terminated with the birth of the malformed baby, there was hydramnios. The pregnancy was full term; the baby, a boy, was born alive, birth weight 2,550 gm., but died immediately after birth. It was recorded that the child breathed and cried before death. Autopsy examination of parts of the body other than the head showed no abnormalities; after the autopsy the head was preserved in formalin, and the present report is based on the examination of this specimen.

The face presents a striking appearance (Figs. 1-4); no external nose is present and no nostrils, but above each eye a proboscis projects horizontally, forward, and medially. The two probosci are very similar in form, the resemblance extending even to minor folds and depressions. Each proboscis is attached in the region of the supraorbital margin, the right one above the midpoint of the upper eyelid and the left just lateral to the midpoint. The diameter at the attachment is 6-7 mm., and from this narrow zone the probosci, each 24 mm. in length, broaden out to a terminal diameter of 11-12 mm. at the free extremity. The latter shows an oval dimple, 3 mm. deep, the lower lip of which is more prominent than the upper. In the floor of this dimple a narrow pit is evident 1.5 mm. in diameter. Neither a delicate probe nor a bristle can be introduced via this pit, although on the right side a small drop of the fixative fluid emerges from the pit on compressing the proboscis. An attempt was made on both sides to inject fluid through the pit into the proboscis with a view to following the course of the fluid thereafter. However, none at all could be introduced. To avoid destruction of the specimen, the probosci were not dissected, nor were histological sections prepared.

The region in which the external nose is normally situated is completely flat. Following this zone downward from the area between the two inner palpebral canthuses it is seen to shelve backward till it reaches the region of the upper lip (Figs. 1 and 2). The upper lip itself protrudes and bunches forward; on its external surface is a central vertical sulcus, very shallow, narrower than a normal philtrum and without the clear margins of a normal philtrum (Fig. 4). The free margin of the upper lip is bowed markedly upward but is otherwise normally formed.

An electric nasopharyngoscope was used to examine the mouth and pharynx. The hard and soft palates and the uvula are normal in appearance; a nasopharynx is present, but no choanae can be seen.

The distance between the two orbits appears to be within normal limits, although on radiological examination (see below) the distance between the medial orbital margins is found to be less than normal. The right eye is large and protruberant. The cornea is completely opaque, but this is probably the result of the formalin fixation; its curvature is not excessive. The ocular conjunctiva is dark brown in color and has a wrinkled appearance; on examination with a lens these wrinkles appear to be due in part to vessels. The corneal margin is not sharp, and vessels seem to be passing from the wrinkled ocular conjunctiva into the peripheral part of the cornea; in the place where

Submitted for publication March 6, 1959.

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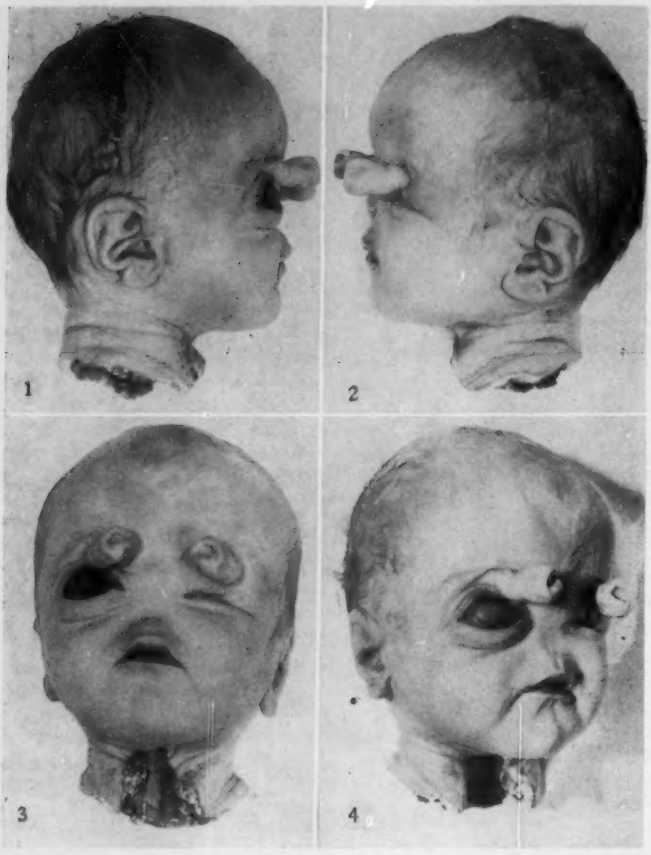


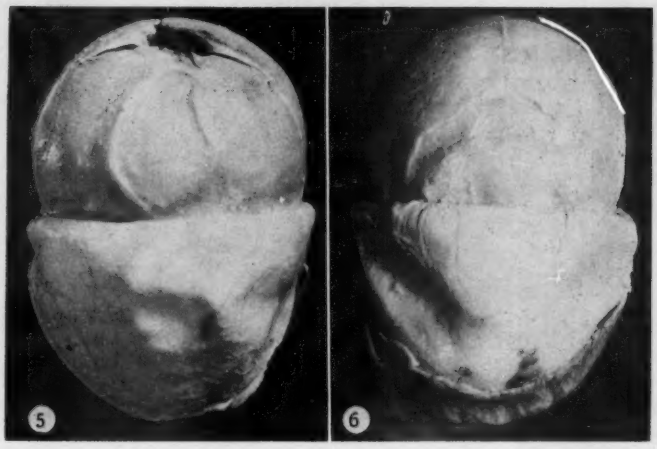
Figs. 1-4.—Various views of the head of the infant with bilateral nasal proboscis. Diffuse pigmentation of the conjunctiva of the right eye is also seen. On the left side there is anophthalmia and coloboma of the upper lid.

these vessels are situated the cornea is also pigmented. The palpebral conjunctiva, as far as this can be seen by slightly everting the eyelids, has the same brown color. No coloboma of the eyelids is evident; the iris and the lens cannot be seen because of the opacity of the cornea. The eyelids are widely separated, the upper lid appearing to be drawn markedly upward (Fig. 4). The lacrymal papilla and punctum of the lower eyelid are present in their normal situation, but neither papilla nor punctum can be seen in the upper lid.

On the left side no trace of an eye can be seen. The eyelids are smaller in all their dimensions than on the right side; they are closed and turned inward toward the orbital cavity. An irregularly notch-shaped coloboma of the upper eyelid is present, located a few milimeters below the attachment of the left proboscis (Fig. 3). When the eyelids are pried apart a shallow cavity is visible, approximately 8 mm. deep; the white, opaque lining of this cavity is continuous with the palpebral conjunctiva. Puncta lacrimalia are present on both lid margins in their normal situation; a papilla is present on the lower lid but not on the upper.

When the scalp was turned downward to reveal the calvaria, two unusual features became evident. First, within the metopic suture there is an additional, circumscribed cranial bone 44×30 mm. in size, like a large sutural (Wormian) bone but with smooth margins (Fig. 5). Second, a shallow sulcus, 1 cm. wide, runs immediately below and parallel with the lambdoid suture (Fig. 6).

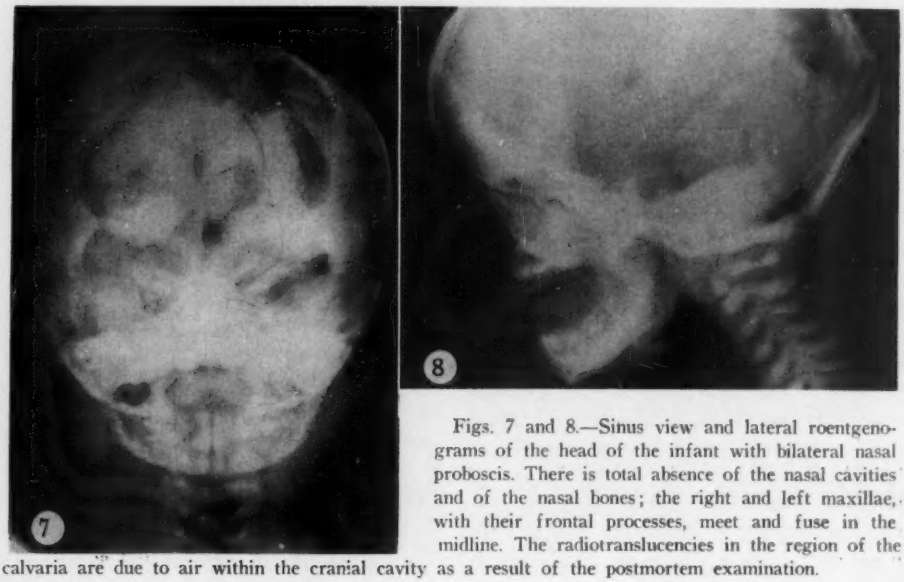
When the brain was removed, it was seen that there is no cribriform plate present; the central part of the middle cranial fossa is much widened in conformity with an unusual, large, centrally placed "additional" lobe of the brain. Olfactory bulbs and tracts are not observed. The arrangement of the dura mater is also abnormal. A description of the dura and the brain will form the subject of a subsequent publication.



Figs. 5 and 6.—Frontal region (Fig. 5) and occiptal region (Fig. 6) of the skull of the infant with bilateral nasal proboscis. The scalp has been turned down. In Figure 5 an additional circumscribed bone (44×30 mm.) is seen within the metopic suture. In Figure 6 a shallow sulcus, 1 cm. wide, is seen to run below and parallel with the lambdoid suture.

Roentgenographic examination (Figs. 7 to 10) revealed total absence of the nasal cavities and of the nasal bones; the right and left maxillae with their frontal processes meet and fuse in the midline, and the distance between the medial orbital margins is less than normal (Figs. 7 and 9). The distance between the prosthion (the midpoint of the anterior edge of the superior alveolar margin) and the dorsum sellae is less than normal (Figs. 8 and 10). The maxillae thus appear to be "displaced" medially and backward. There is no radiological evidence of bone within the probosci. The right orbit is larger than the left (Fig. 7).

Occlusal roentgenograms of the upper jaw show the unerupted buds of the following deciduous teeth on each side: one incisor, one canine, two molars. Judging by the degree of development of the crowns of the incisors it is likely that they are both lateral incisors, but it is impossible to be certain. Control roentgenograms of the lower jaw show a full complement of normally developed deciduous tooth buds.



Figs. 7 and 8.—Sinus view and lateral roentgenograms of the head of the infant with bilateral nasal proboscis. There is total absence of the nasal cavities and of the nasal bones; the right and left maxillae, with their frontal processes, meet and fuse in the midline. The radiotranslucencies in the region of the calvaria are due to air within the cranial cavity as a result of the postmortem examination.

Figs. 9 and 10.—Control sinus view and lateral roentgenograms of the head in normal newborn infants.

Comment

Lateral nasal proboscis is an anomaly presenting a number of interesting embryological problems.

The unilateral probosci which have been described in the literature vary in size and shape. Degree of development of the nose on the same side as the proboscis also varies considerably; it may be almost normally developed (Kirchmayr, 1890), it may be partly formed (Meyer, 1956), or it may be entirely absent so that only the opposite half of the nose is present (Selenkoff, 1884; Tendlau, 1918; Smith, 1950). The embryological origin of the proboscis probably differs somewhat in these various types of cases.

The normal embryological development of the nose commences with the appearance of the nasal placodes; these soon invaginate to form the nasal pits, which are the forerunners of the nasal cavities. The external nose is formed from the medial and lateral nasal processes, which are the respective boundaries of the pits; it is likely that lateral probosci result from malformation of these nasal processes. In those cases where the nose, on the same side as the proboscis, is present to a greater or less extent, the proboscis probably developed from a variable proportion of the lateral nasal process. In cases in which the ipsilateral half-nose is missing entirely, it is likely that the proboscis was derived from both the lateral and the medial nasal processes. This would apply also to the case here described, the maldevelopment having occurred bilaterally. The dimple at the free end of each proboscis probably represents the original olfactory pit which did not go on to form the nasal cavity as it does in normal development. This would account for the absence of the nasal cavities.

Some authors (Grunberg, 1909; Zausch, 1926) are of the opinion that the medial nasal process may be said to have taken part in the formation of a lateral nasal proboscis only if the parts normally formed by the medial nasal process are missing. According to these authors, the structures so formed include the premaxillary bone, and one might have expected, therefore, that in our case all the incisor teeth would have been missing (Patten, 1953). Two alternative interpretations may be offered to explain the fact that this is not so.

1. The premaxilla (globular process) is usually regarded as the lower part of the medial nasal process, but according to Streeter (1948), the premaxillary growth center is distinct from the medial nasal process. It would thus seem possible for defective development of the medial nasal process to occur without the premaxillary center being affected. The latter would then go on to form the central part of the upper jaw, including the incisor teeth and their alveolar processes.

2. Alternatively, it may be postulated that the premaxillary center also enters completely into the formation of the proboscis and that in its absence the maxillary process reaches the midline. The incisor teeth would then be formed from the maxillary process. According to some authors (Frazer, 1953; Woo, 1949), the maxillary processes normally overgrow the premaxillary centers and meet centrally, but this has been denied by others (Noback and Moss, 1953). The subject has been reviewed by Klaff (1956).

The relationship of the lateral proboscis to the condition known as split nose is also of interest. Bischoff (1898) maintained that lateral proboscis could be regarded as a variant of median split nose. In some cases of the latter the two halves of the external nose are indeed almost tube-like in form; such cases have been described and illustrated by Lehmann-Nitsche (1901) and Meyer (1956). A photograph of the latter case is reproduced in Figure 11.

Fig. 11.—A case of hypertelorism with split nose, showing the almost tube-like form of the two halves of the nose. Reproduced with permission from Meyer, R.: *Pract. Oto-rhino-laryng.* 18:399-416 (Nov.) 1956; Basel, S. Karger AG.

As to the cause of the maldevelopment in our case, no explanation can be offered. The history of hydramnios in the mother may be of significance; emphasis has been placed recently on the association between this condition and congenital anomalies in the infants delivered, particularly anomalies of the gastrointestinal and central nervous systems (Lloyd and Clatworthy, 1958; De Young, 1958).

Summary

A case is described of bilateral nasal proboscis with complete absence of the external nose and the nasal cavities. Among the associated anomalies were left-sided anophthalmia, coloboma of the left upper eyelid, diffuse pigmentation of the conjunctiva of the right eye, anomalies of the lacrimal papillae and puncta, and anomalies of the bones of the calvaria and of the brain. The embryological origin of lateral nasal proboscis is discussed, as is its relation to the developmental problem of the premaxilla and to the condition known as split nose.

Dr. E. Levy, Pathologist to the Central Emek Hospital, Affulah, sent us this interesting specimen and provided us with the clinical history. Dr. A. Schwartz and Dr. M. Weinreb furnished the roentgenographs and interpreted them for us. Mrs. E. Salomon and Mrs. H. Weinman supplied the photographs, and Mr. I. Hayoun provided technical assistance. Dr. Rudolf Meyer and Messrs. S. Karger gave permission for reproduction of Figure 11.

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The Use of a Prosthesis in Stapes Mobilization

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Restoration of hearing in otosclerotic deafness depends on being able to reestablish the ossicular conductive mechanism so that air-borne sounds will reach the cochlea. Inability to relieve the stapes ankylosis, which is responsible for the hearing loss, is the reason that mobilization surgery fails. Whether the stapes can be mobilized completely, with both crura and the footplate intact, depends on the degree of fixation and the anatomical structure of the stapes.[1] Occasionally, when the crura fracture, part of or the entire footplate will be simultaneously mobilized, but more often it will remain solidly fixed. In either case, there is usually a separation of the crura from the footplate (Fig. 1). We know that there must be continuity of ossicular transmission to improve hearing but that it is not necessary to free the entire stapes in one piece to accomplish this. A similar result can be obtained in the following situations[2]:

1. When one crus remains in continuity with one-third or more of a mobile footplate. Generally this is the posterior crus.
2. When one or both of the fractured crura make a firm contact with a mobile footplate.

When the crura fracture, efforts to bring them in contact with the footplate are generally of no avail. Should a contact with the footplate be made, its permanence cannot be assured, since it may separate within 24 hours. This is evidently the case when the hearing gain is lost the day after surgery.

The reverse situation may also occur in a small percentage of cases when it is not possible to obtain crural footplate contact in the operating room. An improvement in hearing becomes apparent between the 10th and 14th days as a result of organization and fibrosis in the oval window well, creating continuity between the broken crura and the mobile footplate. However, it is impossible to accurately predict when this situation will occur. The hearing improvement obtained either by contact or through continuity can be equal in degree and cannot be distinguished audiometrically.

Various techniques have been tried in attempts to obtain contact between the crura and a mobile footplate. Absorbable gelatin sponge (Gelfoam) has been placed under the crural arch where it would expand with blood and perilymph and so make contact between the crura and mobile footplate. Improvement in hearing was often spontaneous but would last only 24 to 36 hours.

Another procedure that has been tried in these cases was elongation of the posterior crus after the footplate had been mobilized. In the occasional case, the length of the posterior crus can be increased so that it

Submitted for publication March 10, 1959.

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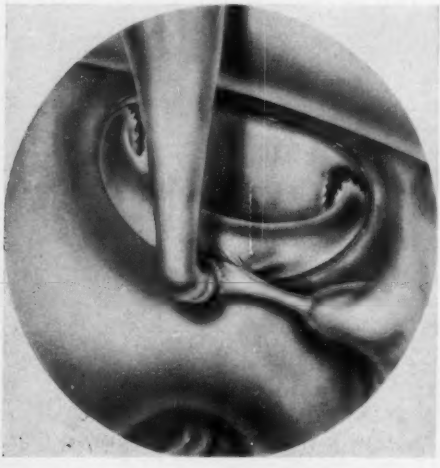
Fig. 1.—Fracture of crura and their separation from the footplate.



will make permanent contact with the footplate. The head of the stapes is disarticulated from the lenticular process and reset under the incus after it is rotated back, dipping the posterior crus down to the footplate and swinging the anterior crus up and out of the well. This elongation technique has only worked in the very infrequent case. Shea's [3] vein-graft technique has not been tried.

It is therefore seen that the management of crural fracture in stapes surgery has really been on a hit-or-miss basis and generally unreliable.

In an effort to establish a more consistent and reliable method of reestablishing conductive continuity following fracture of the stapes crura, the use of a polyethylene prosthesis has been adapted. It is used in place of the broken crura to close the sound-conduction gap between the incus and the footplate after it has been mobilized. It can be used in suitable cases where there is no gain in hearing, whether or not the footplate was simultaneously mobilized at the time of the crural fracture.

Technique

The general technique is as follows: The broken crura and head of the stapes are swung out of the oval window well. The stapedius tendon is cut, and the crura and head of the stapes, which are then loose in the middle ear, are removed. This reveals a direct exposure of the footplate, including its inferior margin, which is rarely visible with the crura intact. The footplate is then accurately mobilized, under direct vision. A piece of polyethylene tubing is set in a vertical position with one end in contact with the footplate and the opposite end wedged under the lenticular process of the incus (Fig. 2). When this technique is executed correctly, in the proper case, it is possible to bring the hearing to a level where the entire air-bone gap is closed. It should be noted that this procedure cannot be used in all cases of crural fracture.

Fig. 2.—Polyethylene tube making contact with the incus and a mobile footplate.

The prerequisites for this technique are as follows:

1. The oval window area must not be contracted down or encroached upon by the adjacent anatomy.
2. The complete footplate should be visible, even if it is involved with extensive otosclerotic disease.

Observations

1. If the footplate is mobilized in one piece, placement of the polyethylene tubing on its posterior half will yield the maximum improvement in hearing. Should the posterior half or three-fourths of the footplate be mobilized, placement of the tube on this portion will give a significant gain.

2. The tubing must be of such a diameter that it does not fit tightly into the well (P. E. 50 is usually used).

3. Polyethylene tubing must not touch the annular ligament or the adjacent promontory. Separation of the footplate from the annular ligament is to be avoided if possible.

4. When the polyethylene tubing is set down on the footplate, its length should be such that its upper end will not slip readily under the lenticular process of the incus. If it does, it is too short and there will not be enough pressure applied to it by the incus. The incus has to be lifted up slightly and set down on the polyethylene tubing with a spring-like effect. This slight pressure is evidently needed to obtain maximum transmission of sound to the cochlea.

5. When the lenticular process is disengaged from the stapes, a small piece of fibrous capsule usually remains attached. It is desirable to let this piece of fibrous capsule sit directly inside the lumen of the

polyethylene tubing when it is in its proper position under the incus. If this capsule is absent, the lenticular process will usually lock itself into the lumen of the tubing if the proper size is used.

6. Once the correct position of the tubing is obtained, it will generally not slip out. However, this has happened, and in certain cases, it is necessary to cut small notches in the upper end of the polyethylene tubing to grasp the incus more firmly.

The required length of the polyethylene tubing will vary from case to case. It is cut with a Bard Parker blade, against the finger. It is slipped over the point of a straight pick and brought down into the middle ear. The tube is disengaged from the pick by catching its free edge under the annular rim. Final positioning is obtained with a curved pick.

If the polyethylene tubing is too short, it must be carefully removed, since it is at this stage that complications may develop as a result of trauma to the membranous labyrinth beneath. The vertical polyethylene tubing may fill with a mixture of perilymph and blood owing to capillary attraction. If the footplate is fractured and partially open, contact of the suction with the open end of the tubing will suck up perilymph as though it were a straw. Once the polyethylene tube is manipulated out of the oval window well, it is easily picked up and removed with a suction tip.

This polyethylene tube technique *cannot* be used in Bellucci[4] Class 4 cases where the promontory and the facial nerve are closely approximated so that the oval window well is very narrow and deep.

It *should not* be used (1) when the footplate fragments into several pieces during its mobilization; (2) when one margin of the footplate becomes free and is depressed toward the labyrinth like a hinged door. In these cases there is the danger of the polyethylene tube being pushed too deeply or falling into the vestibule of the labyrinth.

During five years' experience with stapes-mobilization surgery, cases have been observed before any instrumentation where the incudal stapedial joint capsule was widely spread. Despite the appearance of a closed joint, the lenticular process of the incus was pulled back off of the stapes or the stapes was tilted back in such a position that only a loose membranous contact between the two ossicles existed. In such cases, after the fixed stapes was mobilized, if the poor approximation of the ossicles could not be corrected, there was no gain in hearing. Whenever the incus has a tendency to spring away from a mobile stapes, no improvement is noted. These situations have been corrected and hearing has been improved after purposeful fracture of both crura and their removal along with the head of the stapes followed by implantation of polyethylene tubing of the proper length. A firm approximation can then be made with the tubing between the mobile footplate and the lenticular process and hearing can be improved. This has been done successfully on two occasions.

Complications

1. Severe and prolonged vertigo will result if the tubing is depressed into the labyrinth. After the acute vertigo, subsides, which is due to traumatic disturbance of the membranous labyrinth, postural vertigo will remain for several months.

2. Further deterioration of hearing can result.

Both of the above complications were noted in the same patient early in this series of cases.

Results

The results have been interesting and gratifying to observe. The return of hearing is spontaneous and clinically the same as if a complete intact mobile stapes had been obtained. Improvement in hearing is noted at once when the polyethylene tubing is in its proper position.

The polyethylene tube prosthesis technique was used in a series of 95 patients. Ninety-three of these cases had their crura

fractured during attempts to mobilize the whole stapes. Included in this group are 18 cases where the fracture had been produced during previous surgery, prior to the use of the operating microscope.

Two cases also included in this series had stapes that were completely mobilized, but there was no improvement in hearing because of a widespread incudostapedial joint. Purposeful fracture and implantation of a polyethylene tube gave the desired results in each instance.

Eighty-one of the ninety-three patients where the crura had been unintentionally fractured had a significant result with the polyethylene tube. Therefore, a total of 83 patients in this series of 95 obtained improved hearing.

The air-bone gap was closed in 29 cases.

Forty-two cases came within 20 db. of the bone-conduction level in the major speech frequencies (a gain of 15 to 35 db.).

Twelve patients improved 10 to 15 db. in these same frequencies.

It is too early to accurately determine the long-term results of this prosthesis technique. However, there are reasons to believe that the rate of recurrence may be less than the general over-all rate that has been observed with mobilization surgery during the last five years. In stapes work, we have observed that the lowest rate of recurrence has occurred in those cases where the footplate was fractured adjacent to and not through the otosclerotic pathology, so that one half or more of the footplate was left in continuity with the posterior crus. Now, when it becomes necessary to use the prosthesis technique, since the entire footplate is visualized directly, it can be fractured more accurately behind or adjacent to the otosclerotic area producing ankylosis.

Conclusions

Stapes surgery has gone through an evolutionary process for five years because of the pooling and interchange of ideas that have advanced the surgery to its present state. Yet, despite our newer techniques, fracture of the crura, producing a gap in the sound-conducting mechanism, still occurs in about 25% of cases. Though percentagewise this group has diminished, it is evident that there will always be certain cases in which it will always occur.

To salvage the hearing in this group of cases, a polyethylene tube prosthesis has been successful in reestablishing continuity and restoring hearing when it is placed between a mobile footplate and the incus after removal of the crura and head of the stapes. This technique is not always successful, nor can it be used in all cases when crural fracture occurs during mobilization surgery. However, the improvement in hearing that has been obtained with this procedure in the proper case seems to indicate that its use has a place in stapes surgery.

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Amyloid Tumors of the Larynx and Trachea

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Amyloidosis is the deposition into tissues of a substance which Virchow called amyloid because of its resemblance to starch. It is a mucopolysaccharide-protein complex which changes the color of iodine as starch changes it, and it is specific in its absorption of Congo red and methyl violet. According to Peters,[1] the substance is deposited in extracellular spaces, forming on tissues as a product of a disturbed protein metabolism of the cellular membranes. Larsen[2] has suggested that amyloid forms on tissues because of a change in the capillary permeability not associated with any alteration of tissues.

Many theories have been developed in an attempt to determine the etiology of amyloidosis. Amyloid deposits have been considered the product of a disturbance of protein metabolism, the result of an antigen-antibody reaction, and the consequence of a reticuloendothelial disturbance, and, in those cases associated with multiple myeloma, it has been suggested that the hyperproteinemia has been a related cause. Clinically, four forms of amyloidosis are recognized:

1. Typical or secondary amyloidosis: amyloidosis which follows a chronic infection such as pulmonary tuberculosis with associated suppuration, chronic osteomyelitis, etc.

2. Atypical or primary amyloidosis: amyloidosis which develops without relationship to other disease.

3. Amyloidosis which develops in association with multiple myeloma.

4. Tumor-like amyloid formation. This form is relatively rarer than the first three; tumors of this type are found most frequently in the respiratory and urinary systems.

The first case of amyloid tumor of the larynx was reported by Borow and Neumann, in 1875. Since that time, additional cases have been reported by New, Pollak, Kramer, Som, and others. A thorough review of the literature is included in the report by Kramer and Som.[6] Schottenfeld et al.[7] in an analysis of 14 cases of amyloid tumor of the respiratory tract, found that 6 involved the trachea and larynx, 2 of which extended into the bronchi. According to Stark and New,[4] a distinction must be made between those cases in which the amyloid deposits of the larynx and trachea are part of a generalized amyloidosis and those in which the deposition of amyloid is localized with no evidence of a generalized disease. The localized amyloid deposits in the larynx and trachea can be divided into three groups; degeneration of a previous tumor, isolated tumor formation, and diffuse subepithelial infiltration.

Many causes of amyloidosis have been suggested. If the existence of a previous chronic infection is considered the etiologic agent, the infection should be limited to the area involved. However, in most examples in this category, such infection has not been proven. Spain and Barrett[8] called attention to the existence of these tumors in areas of cartilage and suggested the possibility of previous enchondrosis or exostosis as a causative factor.

Submitted for publication March 16, 1959.

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Read in part, in Spanish, before the VI National Meeting of Otolaryngology, Guanajuato, Mexico, May, 1956, and published in Spanish without illustrations in the *Anales de la Sociedad mexicana de otorinolaringología* May, 1956, Vol. 6, No. 29.

Clinical Picture

Amyloid tumors of the larynx and tracheobronchial tree are rare. The recognition of primary amyloid disease in the respiratory system usually follows investigation of hoarseness or signs of respiratory obstruction, and the diagnosis is made on microscopic examination of the tissue removed after endoscopic biopsy.

According to the literature, most of the cases occur after the fifth decade of life, although Stark and New[4] reported an amyloid tumor in a patient 19 years old. Amyloid tumors of the larynx are more frequent in males than in females, in the ratio of 3:1. In the group reported in this paper the females outnumber the males 5:2.

The symptomatology is not characteristic. The type and degree of symptoms will depend upon the location and size of the mass; in some instances, the presence of the tumor has been an incidental finding at autopsy.

Pathology

Amyloid tumors usually appear as a granular, waxy, transluscent, yellowish or reddish mass without ulceration. Microscopically, there appears to be a subepithelial infiltration of a homogenous hyaline substance forming concentric layers with giant-cell infiltration. Some authors describe an involvement of the capillaries with this type of infiltration, and others describe the involvement of the mucous glands with replacement of the glands by amyloid masses.

Review of Cases

Case 1.—A 35-year-old white woman had a history of persistent hoarseness of two years' duration. There was slight shortness of breath but no dysphagia or cough. The patient appeared to be in good health, and general examination failed to reveal any other pathology. Mirror examination of the larynx showed a swollen right false cord, almost suggesting the appearance of a laryngocele. The left cord was normal, and its motility was normal. The right true cord was covered by a soft, red, granular mass, and the left true cord was seen to slide under this mass on phonation. On direct laryngoscopy, the tense, swollen appearance of the right false cord was observed, and when this structure was retracted laterally, verrucous tissue on the superior surface of the right true cord was exposed. The tissue was grasped with bent cup forceps and was found to be extremely friable. It was removed, leaving a relatively normal superior surface of the right true cord. The lesion appeared to have its origin in the right laryngeal ventricle. Histologic examination proved this to be amyloid deposits in the larynx. The patient's voice improved after the operation, and the improvement has been maintained for two years. However, there has been some increase in the overhanging right false cord.

Case 2.—A 51-year-old white man complained of hoarseness of one year's duration. There was no dyspnea or dysphagia. A mirror laryngoscopy showed a smooth swelling of the right false cord which completely covered the right true cord. The chest x-ray revealed a narrowing of the trachea on the left, and a planogram of the neck showed the presence of a mass in the right laryngeal ventricle (Fig. 1). On direct examination of the larynx, the entire right side was found to have lost its normal configuration. The right false cord, ventricle, and right true cord were swollen and protruded into the glottic lumen. The true and false cords were indistinguishable from each other, and the ventricle was entirely obliterated. The mass had a firm consistency, and needle aspiration yielded no fluid. A tracheoscope was introduced because of the compression of the left side of the trachea. As the smooth tip of the instrument reflected the mass in the larynx, the walls of the trachea as far as the carina were found to be studded with white, glass-hard, granular spicules, protruding most prominently from the left. Separate pieces of tissue were removed from the trachea and the larynx. The former proved to be osseous material confirming the diagnosis of tracheopathia osteoplastica; the tissue from the larynx was reported histologically to show edematous and hyalinized fibrous laryngeal mucosa.

Because of the possibility of an underlying laryngocele, surgical exploration was done, approaching the mass over the right thyroid ala. The mass was found to be granular and firm, and further exposure necessitated a thyrotomy. The entire mass was excised, and a frozen section indicated the lesion to be amyloid infiltration. A tracheotomy was done after the removal of the mass, and the patient made an uneventful recovery and was extubated the sixth postoperative day. Sections of the specimen were stained with Congo red, showing the characteristic coloration of the amyloid substance. The voice improved after the resection, but six months later moderate hoarseness recurred, and a small mass of granulation tissue was found in the anterior commissure. This was

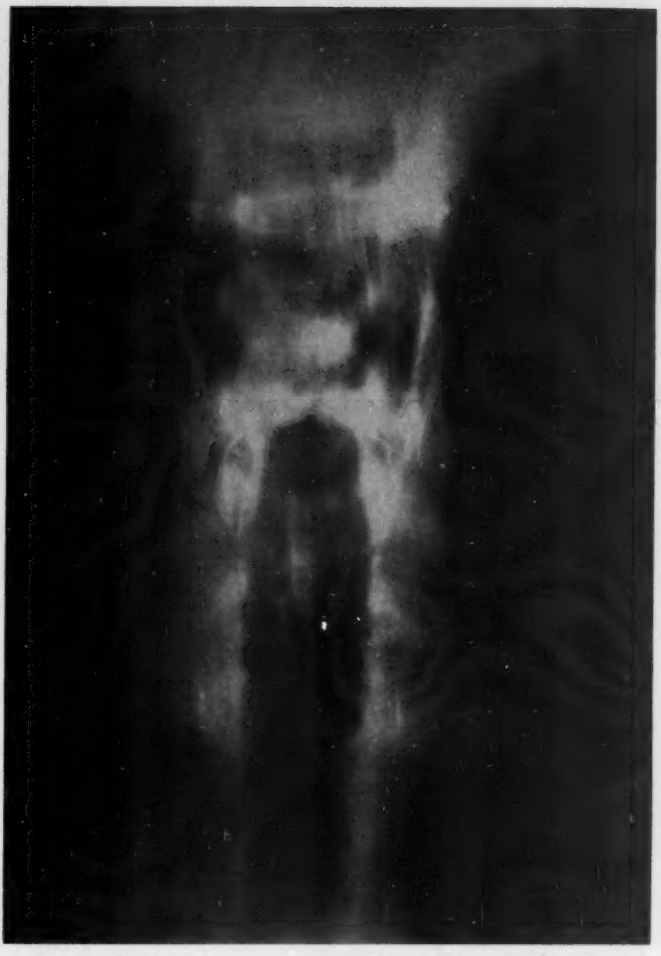

Fig. 1 (Case 2).—Planogram, showing amyloid tumor involving the right false cord. It was resected through a thyrotomy. The irregularity of the tracheal walls is due to a tracheopathia osteoplastica, an incidental finding.

removed under direct laryngoscopy. The sections showed no amyloid infiltration, consisting only of nonspecific granulation tissues. The right ventricle was clear at this time.

CASE 3.—A 60-year-old white woman was admitted complaining of hoarseness of six months' duration. The patient's general condition was good, and a careful medical examination revealed no significant pathology. Indirect examination of the larynx revealed a marked thickening of both ventricular bands and a granular mass in the anterior commissure, producing a lateral displacement of the vocal cords. The motility of the cords was reasonably good. Direct laryngoscopy showed a soft, buttery mass in the anterior commissure extending posteriorly along the ventricular bands. The section demonstrated the presence of amyloid deposits. A second laryngoscopy, performed after the intravenous injection of Congo red, showed the mass to have a reddish appearance, with an extension posteriorly and inferiorly as far as the upper part of the posterior wall of the trachea. All redundant tissue was removed endoscopically, and immediately thereafter the patient's symptoms improved. Examination five months later revealed no recurrence of the mass, and the patient's voice remained clear.

CASE 4.—A 53-year-old white woman had had hoarseness for more than a year, not associated with dyspnea or dysphagia. Her general health had been good. Mirror laryngoscopy showed a granular, mulberry-like lesion on the posterior third of the right vocal cord. On direct laryngoscopy, the mass was seen to originate on the upper surface of the right vocal cord and extend into the right laryngeal ventricle. The mass was re-

moved with cup forceps and the cord edge straightened. Histologic examination of the tissue demonstrated a narrow edge of stratified squamous epithelium, covering hyaline material in irregular large and small single and confluent masses with rounded edges. In the interstices between the masses were infiltrations of lymphocytes and plasma cells. At some levels along the edge of the hyaline material were foreign-body giant cells. There was also a small amount of lymphoid tissue. The flattened squamous epithelium at the margins was continuous with a pseudostratified columnar layer. The hyaline material in masses, single and confluent, had the appearance of amyloid. The hoarseness disappeared, and three months later a mirror laryngoscopy revealed a normal larynx.

CASE 5.—A 75-year-old white man gave a history of gradually increasing hoarseness, inspiratory stridor, and dyspnea for approximately one year. Otherwise, the history and general physical examination were essentially negative. Increased vascularity, thickening of the vocal cords, and the presence of a yellowish-red tissue below both cords was found on mirror laryngoscopy. The chest x-ray showed no evidence of pulmonary disease, but planographic studies of the neck disclosed a subglottic stenosis (Fig. 2). Direct laryngoscopy showed that this mass involved the entire conus elasticus, beginning immediately at the phonating level of the cords and extending downward into the subglottic area (Fig. 3). The tissues were soft, pinkish-yellow in color, irregular, and nodular in appearance. The subglottic space was markedly narrowed by the encroachment on the lumen of this tissue, the tissue protruding more from the left side than from the right. The motility of

Fig. 2 (Case 5).—Planogram of the neck, showing bilateral subglottic swelling due to amyloid tissues.

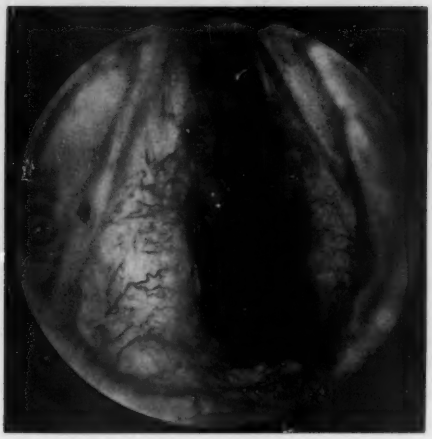

Fig. 3 (Case 5).—Direct laryngoscopic photograph of the larynx seen on planography in Figure 2. Smooth, nodular masses with a superficial vascular pattern are seen extending subglottically from the level of the true cords.

the cords was fair. Histologic examination of the tissues demonstrated a large amount of hyaline material through which narrow crevices and capillaries extended, separating the hyaline material into masses. Scattered along the crevices were a few foreign-body giant cells. The surface was a narrow layer of stratified squamous epithelium. There were also compressed crevices lined by pseudostratified columnar epithelium. The structure of this hyaline material in the stroma corresponded to amyloid deposits. Subsequent direct laryngoscopy performed after the intravenous injection of Congo red failed to reveal any change in the appearance of the mass. Further endoscopic removal of tumor tissue was done to restore the airway to its normal size, with alleviation of dyspnea and improvement in the voice.

Case 6.—A 74-year-old man was admitted as an emergency with severe dyspnea, cyanosis, and the appearance of desperate air hunger. He was irrational. He had a history of choking of two years' duration, associated with cough and attacks of expiratory dyspnea which had been diagnosed as bronchial asthma and treated with epinephrine. The frequency and intensity of attacks had increased, especially during the three weeks prior to admission. On admission, because of the acute respiratory obstruction, the patient was immediately taken to the operating room, where a bronchoscope was introduced, revealing an obstructing mass in the trachea. The bronchoscope was forced through the mass into the left main bronchus, relieving the respiratory obstruction. A piece of tissue was cored out of the tumor by this procedure and sent to the laboratory, and the patient was returned to his room breathing easily. Histologic examination demonstrated the tissues to be composed of hyaline material. In this background of hyaline material were dilated blood channels, and around some of them were small infiltrations of lymphocytes and plasma cells. While at some levels the hyaline material occupied all portions of the stroma and seemed to be a continuous mass, at other levels there were smaller aggregates with interstices between them. The individual aggregates varied considerably in size and extended directly to the surface epithelium, some of which was a thin layer of stratified squamous and some pseudostratified and thickened but resembling the lining of the trachea. The hyaline material extended directly to the basal edge, except for small infiltrations of lymphocytes. The amount of hyaline material removed was large, and the sections removed consisted entirely of this substance with the typical waxy appearance of amyloid deposits in the mucosa of the trachea. There were no cancerous tissues. Five days after the initial bronchoscopy, following an intravenous injection of Congo red, a second bronchoscopy showed the amyloid material extending from below the larynx to the carina, with the greater portion of the mass in the midtrachea. The mass was granular and reddish in appearance. Additional large pieces of tumor were removed. The histologic report of the tissues demonstrated "amyloid deposits in the lining of the trachea" in which a red coloration was found. The patient was seen three months later with no evidence of breathing difficulty. Two years later symptoms of obstruction appeared, and at bronchoscopy gross amyloid tissue was again removed. Subsequent examination, four years after the initial bronchoscopy, when the patient was 78 years of age, showed no recurrence of the tumor.

Case 7.—This 48-year-old woman had hemoptysis for several days in 1942 following severe coughing; the patient was symptom-free until 1948, when she developed productive cough, fever, and right subscapular pain. X-rays showed an area of atelectasis on the right upper lobe. Examination of the sputum was reported as showing an occasional acid-fast organism, and a tentative diagnosis of tuberculosis was made. Bronchoscopy at that time revealed an irregular nonulcerative lesion around the right upper lobe orifice, and a biopsy specimen of the material was found to contain amyloid deposits. Sanatorium care was advised, but the patient remained at home without treatment. Two years later she became dyspneic on exertion and developed symptoms of bronchial asthma. She was admitted to the hospital for bronchoscopy, and at this examination the tracheal walls were found to be thickened, nodular, and red. There was considerable obstruction of the

trachea and right main bronchus, and the orifice of the right upper lobe bronchus was occluded. Several bronchoscopic procedures were done, with removal of soft obstructing tissue to clear the tumor from the lower trachea and the right main bronchus. Histologic examination proved the tissue removed to be amyloid. Subsequent examination in 1951 showed no recurrence of the obstruction, although bronchoscopic examination in April, 1956, showed a thickening of the tissue lining the trachea and both main bronchi. Only minimal nodularity was present, causing no clinical manifestations of tracheal or bronchial obstruction. X-ray shows the atelectasis previously present in the right upper lobe to have cleared.

Diagnosis

A positive diagnosis of amyloid tumor is made on the microscopic examination of the tissue, sectioned and stained in the routine manner, e. g., hematoxylin and eosin. A regular mucosal surface encompasses rounded masses of varying size. The masses are composed of hyaline material, in the interstices of which lymphocytes and plasma cells may be present.

Grossly, the tissue has a red-tinged, grayish-yellow color, with a waxy translucent surface. In those instances where Congo red is injected intravenously prior to visual examination, the material can be seen to have taken on a corresponding red coloration. The limitations of the tumor can thus be more easily determined. The microscopic sections do not indicate that Congo red dye has been previously injected.

The Congo red absorption is usually unaltered in primary localized amyloidosis, since the amount of amyloid tissue in these instances is fairly small as compared with secondary amyloidosis.

Prognosis

No instances of maligant change in amyloid tumor of the larynx and trachea have been reported in the literature. The prognosis is therefore entirely dependent on the situation and extent of the mass. Owing to the fact that the tumor is in the airway, the degree of respiratory obstruction determines the prognosis.

Treatment

Since the number of reported cases is small, evaluation of the various suggested methods of therapy is difficult. Endoscopic removal of the amyloid tissue appears to be, at the present time, the treatment of choice. It has been suggested that a partial removal be followed by irradiation, although the rationale of such therapy is open to question. Figi [9] proposes that the resection of the tumor should be followed by the application of a cutaneous split-thickness graft to replace the removed mucosa. Pollock [5] reported cases of recurrence of the lesion after partial removal.

At the present time, the most satisfactory treatment is surgical removal of the amyloid deposits. In most instances, endoscopic removal is adequate, particularly when the tumor is localized to a small area. If the lesion is diffuse, endoscopic removal should be done, taking as much of the tissue as can be obtained with cup forceps or biopsy forceps. Large areas of disease may have to be excised and the cut surfaces replaced with a skin graft, as reported by Figi [9] and referred to by Stark and New.[4]

The disease occurs most commonly after the fourth decade of life, but in the group studied two were under 50 years of age at the time of diagnosis. The prognosis depends on the site and extent of the amyloid deposits. Surgical removal is not as a rule followed by recurrence. No associated general symptoms are noted, and the disease does not primarily shorten the expected life span.

Summary

Seven cases of primary amyloid tumors of the larynx, trachea, and bronchi are reported. Four cases were confined to the larynx, and one of these patients had an associated tracheopathia osteoplastica. One case was chiefly laryngeal, with extension to the upper posterior tracheal wall. In two patients the disease occurred in the trachea and main bronchi. Endoscopic removal was performed in all but one with good results.

In one patient with tracheal involvement the tumor recurred only once and was removed endoscopically a second time with no further recurrence. In one patient a laryngeal tumor was resected through a laryngofissure with a good ultimate result except for slight residual hoarseness.

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The Treatment of Recent Bell's Palsy on a Rational Etiological Basis

Results of Cervical Sympathetic Block and Corticosteroid Therapy

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The name of Sir Charles Bell has been intimately linked with facial palsy since the publication of his collected papers on the anatomy and pathology of the seventh cranial nerve, in 1844. Today, the term "Bell's palsy" is confined to "idiopathic" peripheral facial paralysis, thus excluding cases due to geniculate herpes and other known causes. A large number of cases remain in which the etiology is uncertain and unproved; these are the most frequent, as shown by Park and Watkins (1949), and by Cawthorne (1953).

The last three decades have witnessed an outburst of research into the many problems associated with "idiopathic" facial palsy which is without parallel since Bell's original observations. In particular, special regard has been paid to possible causative factors and attempts have been made to lessen the incidence of permanent cosmetic disability. It is well known that about 80% of all patients regain full function without specific intervention. In 1932, Ballance and Duel advocated surgical decompression for the 20% of cases which failed to recover completely, and in 1947, Kettel propounded the ischemic theory which some present-day otologists endorse (Hilger, 1949; Sullivan, 1950; Cawthorne, 1951, and Korkis, 1957 and 1958).

Since the pioneer reports, surgical attention has been concentrated mainly on the *alleviation* of long-standing paralysis by decompression. The present investigation is concerned with the *prevention* of long-standing paralysis by specific relief of ischemia.

Submitted for publication March 28, 1959.

Delivered by invitation to the Section on Otolaryngology of the College of Physicians of Philadelphia, Jan. 21, 1959.

Neurovascular Theory

According to the ischemic theory, Bell's palsy results from interference with the blood supply to the facial nerve. This takes place through the minute vasa nervorum, which reach the seventh nerve in the unyielding bony Fallopian canal in the temporal bone near the stylomastoid foramen. Ischemia of the nerve leads to ischemic paralysis. The ischemic nerve swells, and compression occurs as a secondary factor. The actual cause of the vascular dysfunction is unknown, but it results in either vascular spasm, intra-luminal thrombosis, or a combination of primary spasm and secondary thrombosis.

The neurovascular theory offers the best explanation of the well-known clinical features and course of Bell's palsy. It explains the sudden onset of the condition, the lack of general constitutional disturbance, the variability in the degree of severity of the paralysis, the differences in the time and degree of recovery, the possible precipitation by exposure to draft, the progression which is sometimes seen from an incomplete to a complete paralysis (secondary compression), and the possibility of recurrence. Mild cases are compatible with minor degrees of temporary spasm and severe ones with long-lasting spasm, or thrombosis.

Thus, the extent and time of duration of the ischemia govern the clinical course primarily, while the degree and duration of the resultant swelling influence the prognosis secondarily.

The results of treatment obtained in the present investigation lend support to the suppositions contained in the neurovascular theory. If spasm is relieved soon after the onset of the paralysis, recovery may begin almost at once in the vasospastic group. In the case of long-continued vasospasm or of primary or secondary thrombosis, quick recovery is not seen; then vasodilator therapy may fail, and it is necessary to relieve the compression either by medical or by surgical means.

Correlation of Etiology with Treatment

Generations of practitioners have treated Bell's palsy as a condition demanding no immediate active measures apart from reassurance, support of the paralyzed musculature, and physical therapy to maintain muscle tone. Such an expectant outlook is outmoded today in the light of the neurovascular hypothesis.

When loss of function is incomplete (i. e., a paresis), complete recovery is the rule. There is no problem in this type of case. When loss is complete (i. e., true paralysis) however, there is no way of determining *in the vital early stages* whether a given case will recover full function (80% of all cases) or whether varying degrees of residual paralysis will persist (20%). Therefore *every* case must be treated as soon as possible by a form of therapy that can be promptly, safely, and easily applied. Surgical decompression on such a wide scale is not justifiable, although, on theoretical grounds, the sooner the operation is done the better.

A rational treatment plan aims at the rapid relief of angiospasm, the prevention of secondary thrombosis, and the reduction of swelling.

Spasm can be treated medically by the use of vasodilator drugs, such as nicotinic acid, but such therapy is uncertain unless large doses are used and is slow unless the intravenous route is employed. Skinner uses histamine injections and Martin employs intravenous procaine with nicotinic acid by mouth for this purpose.

Rapid vasodilatation is brought about by paralysis of the cervical sympathetic system by a stellate block. This can be accomplished by injecting a 1% solution of procaine into the vicinity of the ganglion; of the alternative routes, I prefer the anterior one, which is easy and safe. A flush of the homolateral tympanic membrane and the onset of a Horner's syndrome indicate a successful injection. These easily visible signs, confined to be anatomical area where vasodilatation is required, give immediate proof of the effectiveness of sympathetic paralysis. Swan used this method with success in four cases as long ago as 1952.

Cortisone is known to be capable of reducing tissue edema in the absence of definite infection. With this aim in view, it has been used in the treatment of Bell's palsy by Rothendler and by Robison and Moss, among others. Cortisone itself may be used, but nowadays I prefer the synthetic corticosteroid prednisone because of its lack of undesirable side-reactions.

Material

Early cases of Bell's palsy have been treated by stellate blocks, repeated at two- to three-day intervals, if necessary. In later cases, cortisone has been used either in combination with cervical sympathetic blocks or as the sole therapeutic agent. Unselected cases have been used as controls in this investigation; these include patients with herpes oticus and with straightforward Bell's palsy, treated along orthodox lines by physical therapy, heat, and active exercises.

Table 1 summarizes 65 cases of atraumatic facial palsy which have been adequately followed up for the purpose of this investigation. The seven cases of geniculate herpes are included in this total because they were referred as "Bell's palsy," although subsequent investigation and clinical signs showed their true nature. They form a small, but important, group of controls, as their response to specific treatment by paralysis of the cervical sympathetic system differs from that of vasospastic Bell's palsy cases.

The total shows that there is neither side nor sex predominance. The age distribution of the patients is shown in Table 2. The oldest patients were 71 years of age in the Bell's palsy cases treated specifically, 62 years in the "control" Bell's group, and 73 years in the herpetic variety: the youngest patients were 10 years, 9 years, and 18

TABLE 1.—*Material on Sixty-Five Cases*

	Sex		Side Affected		Total
	Male	Female	Left	Right	
Bell's palsy treated specifically (block &/or cortisone)	17	18	13	22	35
Bell's palsy controls (physical therapy only, or no treatment)	12	11	15	8	23
Herpes oticus	3	4	6	1	7
Total	32	33	34	31	65

years of age in these three groups, respectively. The patients are fairly evenly distributed throughout the various age decades in all three groups (Table 2).

Results of Treatment

1. *Incomplete Facial Palsy.*—As the prognosis differs greatly in complete and incomplete types of facial palsy, it is essential to consider each of these separately. Table 3 shows the distribution of complete and incomplete cases in the three groups. Although incomplete loss of facial function is associated with early spontaneous recovery, the six incomplete "treated" cases were given stellate blocks, one to four in number, at varying times after the onset of the paresis, in an attempt to prove or disprove the vasospastic theory. If relief of angiospasm speeded recovery materially, this hypothesis would be strongly substantiated. The outcome of this investigation is tabulated in Table 4.

TABLE 2.—*Age Distribution in Sixty-Five Patients*

Age, Yr.	Bell's Treated Specifically (35)	Control Bell's (23)	Herpes Oticus (7)
Under 20	4	4	1
21-30	8	3	1
31-40	6	4	1
41-50	6	4	1
51-60	6	5	1
Over 60	5	3	2

TABLE 3.—*Distribution of Complete and Incomplete Types*

Facial Palsy	"Treated Bell's"	"Control" Bell's	Herpes Oticus
Complete	29	22	6
Incomplete	6	1	1
Total	35	23	7

The immediate increase in power of the affected facial musculature within a few minutes of the sympathetic block was most remarkable in all cases, thus affording striking confirmation of the correctness of the neurovascular theory. The average time for full recovery (including emotional balance) was only 4 days from the date of the first injection and 10.7 days from the time of onset of the paresis. Unfortunately, there are not a sufficient number of cases or controls in this series to draw a definite conclusion concerning the effectiveness of the method in speeding full restoration of

TABLE 4.—*Incomplete Facial Palsy Treated by Block*

Case	Time Lapse Between Onset & Treatment, Days	Blocks, No.	Immediate Result	Full Recovery	
				Days After Block	Days from Onset
1	9	2	Improved; 50% better in 24 hr.	2	11
2	5	1	Improved; muscle tone stronger	7	12
3	4	4	Improved	6	10
4	2	1	Improved; marked 2 days later	6	8
5	12	1	Improved	1	13
6	8	1	Improved	2	10

function, but the "control" incomplete case took 14 days and the herpetic one 7 days to recover. Judging from my own previous experience, the impression is that recovery is speeded, as two to three weeks usually elapse before this occurs spontaneously—but my memory may be fallacious.

There is a considerable time lag between the time of onset of the paresis and the first attendance of the patient. The most rapid full recovery was in eight days in a patient treated soonest after the onset (two days). This underlines the importance of relieving angiospasm as soon as possible. Had the injections been given within a few hours, the results might have been more decisive and dramatic. Further investigation of early cases is indicated, as it is important to establish the relationship of the rate of recovery to the duration of angiospasm.

2. *"Complete" Bell's Palsy.*—The 29 treated cases fall into three groups, as follows:

(1) Those treated by stellate blocks alone—20 cases.

(2) Those treated by corticosteroid therapy alone—4 cases.

(3) Those treated by a combination of (1) and (2)—5 cases.

Group 1: Table 5 analyzes the results achieved in patients treated by cervical sympathetic blocks. By full recovery is meant full voluntary motor power with complete restoration of emotional balance. It will be noted that 2 patients attained this within one week; 11, within two weeks, and 3, within three weeks. Thus, 16 out of the 20 (80%) were fully recovered within 21 days; another 3 recovered in 4 weeks, 7 weeks, and 9 weeks, respectively, while after 4 months only 1 has failed to recover completely (5%).

It is worth noting that this one patient was not seen and treated until 23 days after the onset of the paralysis; this long time lag may explain the failure, although another patient who was not seen until the 35th day fully recovered 10 days after the first stellate block. It is probable that there was a greater degree of secondary compression in the case which failed; corticosteroid therapy might have helped but was not given, as slight recovery was noted at the end of two weeks. In the light of present experience, cortisone would have been given if there had been no marked evidence of returning function within a period of three weeks. Moreover, decompression should

TABLE 5.—*Complete Bell's Palsy Treated by Stellate Blocks*

Case	Time Lapse, Days	Blocks Performed, No.	Improvement, Immediate Degree	Recovery	
				Amount	Time, Days
1	2	3	Slight	Full	8
2	1	1	Slight	90%	4
				Full	11
3	5	2	None	Full	6
4	1	3	None	Full	14
5	1	3	Slight	Full	9
6	10	1	Marked	Full	14
7	3	3	Marked	Full	16
8	4	3	Slight	Full	42
9	23	2	None	Incomplete	4 mo.
10	2	4	Marked	Full	4 wk.
11	1	2	Marked	Full	9
12	14	4	Slight	Full	15
13	5	1	Slight	Full	3
14	1	3	Slight	Full	14
15	35	3	Marked	Full	10
16	1	4	Slight	Full	10
17	4	2	Slight	Full	11
18	5	1	Slight	Full	14
19	1	2	Slight	Full	18
20	3	3	None	Full	9 wk.

TABLE 6.—*Control Cases of Bell's Palsy—Complete and Incomplete*

Case	Type of Facial Palsy	Period of Physical Therapy	Recovery	
			Amount	Time
1	Complete	Still under treatment	Incomplete	2 yr. +
2	Complete	Still under treatment	Incomplete	2 yr. +
3	Complete	10 wk.	Complete	4 mo.
4	Complete	10 days	Complete	10 days
5	Complete	15 wk.	Complete	4 mo.
6	Complete	6 mo.	Complete	7 mo.
7	Complete	20 days	Complete	20 days
8	Complete	3 mo.	Complete	3 mo.
9	Complete	10 wk.	Complete	3 mo.
10	Complete	23 days	Complete	23 days
11	Complete	11 days	Complete	11 days
12	Complete	13 days	Complete	13 days
13	Complete	8 mo.	Complete	8 mo.
14	Complete	10 days	Complete	10 days
15	Complete	2 yr.; treatment disc.	Incomplete	2 yr.
16	Complete	6 wk.	Complete	6 wk.
17	Complete	3 mo.	Complete	3 mo.
18	Complete	Still under treatment	Incomplete	1 yr.
19	Complete	10 mo.	Complete	10 mo.
20	Complete	2 mo.	Complete	2 mo.
21	Originally complete	Nil advised	Incomplete	17 yr. later
22	Originally complete	Nil advised	Incomplete	25 yr. later
23	Incomplete	Nil	Complete	14 days

have been performed at the end of six to eight weeks in this case, as steady, progressive recovery was not maintained. In the main, however, these excellent results compare more than favorably with my own past experience and with the control cases in this series treated by conventional conservative measures (Table 6). These can be divided into three types.

(1) Early recovery within four weeks. It is probable that this type would have recovered spontaneously without physical therapy—7 out of 23 (approximately 30.4%).

(2) Intermediate variety. Full recovery in under one year—10 out of 23 (approximately 43.5%).

(3) Late. Incomplete recovery in over one year. Various degrees of residual paresis exist, and contracture from overtreatment by electrical stimulation may cause additional cosmetic imbalance—6 out of 23 (approximately 26%).

What, then, does relief of angiospasm accomplish? It converts the "intermediate" type of case into the early-recovery variety, and it reduces, very materially, the number of patients who are left with residual paresis—surely a worth-while gain to patient, doctor, physical therapist, and the economy of the hospital service.

Group 2: Only four patients have been treated by corticosteroid therapy alone (Table 7).

The number of cases is too small to warrant any worth-while conclusions. Two of the patients refused stellate blocks, and two were being used as "controls." Treatment was instituted later in an attempt to prevent permanent disability when spontaneous recovery did not occur or when recovery became arrested.

Group 3: These five cases fall into two groups: (a) two treated by primary combined therapy, i. e., block and corticosteroid therapy started at the time of the first consultation and (b) three treated by secondary combined therapy, i. e., primary blocks with secondary corticosteroid therapy, commenced at a later date, when recovery was incomplete (Table 8). In case 5, decompression was performed 8 weeks after the onset

TABLE 7.—*Corticosteroid Therapy in Complete Bell's Palsy*

Case	Lapse Between Onset & Attendance, Days	Time After Onset Treat. Begun	Drugs & Dosage	Results
1	1	12 days	Prednisone, 105 mg. in 1/52	Incomplete recovery 85% in 4/12 (block refused)
2	2	28 days	Prednisone, 140 mg. in 12 days	No spontaneous recovery in 4/52; rapid recovery after prednisone for 1/52; complete recovery 4/52 after treatment started (was being used as "control")
3	6	6 days	Cortisone, 25 mg. b. d. for 5 days	Complete recovery 6 days
4	4	16 wk.	Prednisone, 140 mg. in 14 days	Spontaneous recovery began in 13 wk. before treatment started; incomplete recovery in 7/12 (patient refused stellate block)

TABLE 8.—*Complete Bell's Palsy Treated by Combined Methods*

Case	Time Seen, Days	Primary Treatment	Prednisone Dosage	Recovery	
				Amount	Time
1	2	3 blocks + corticosteroids	70 mg., in 7 days	Full	14 days
2	8	1 block + corticosteroids	160 mg., in 14 days	Full	21 days
3	2	1 block without improvement	105 mg., in 14 days; commenced 7 days after block	Full	21 days
4	11	2 blocks with improvement	180 mg., in 28 days; commenced 21 days after block	Full	4 mo.
5	8	5 blocks without improvement	175 mg., in 14 days	No improvement; decompression 2/12 after onset; no return function	

of the facial palsy. At operation, the nerve was found to be atrophic, being converted into a thin thread of fibrous tissue. This patient was 67 years of age and had marked generalized arteriosclerosis. Occlusion of the vessel by intraluminal thickening or thrombosis probably accounts for this failure.

mediate, and late. Cases not yet fully recovered have been classified in the late group. When stellate blocks are employed, either as the sole treatment or in combination with cortisone, 80% of the cases recover very quickly, compared with only 30% in the "control" group.

TABLE 9.—*End-Results of Stellate Blocks in Complete Bell's Palsy*

Type of Recovery	(*a*) Block Alone (20)	(*b*) Combined Block + C-S Therapy (5)	All Cases Treated by Block = (*a*) & (*b*) (25)	Controls
1 Early recovery (under 4 wk.)	17/20 (85%)	3/5	20/25 (80%)	7/23 (30%)
2 Intermediate recovery (under 1 yr.)	3/20 (15%)	1/5	4/25 (16%)	10/23 (43%)
3 Late or incomplete recovery (over 1 yr.)	Nil	1/5	1/25 (4%)	6/23 (26%)

This is a recent case, and there is as yet no evidence of regeneration. As there is no way of determining the type of vascular occlusion, it is felt that a trial of combined treatment was justified before resorting to surgical decompression.

The end-results of vasodilator therapy are summarized and compared in Table 9, the cases being divided into the three groups of early recovery (within four weeks), inter-

3. *Herpes Oticus Cases.*—Unlike Bell's palsy, geniculate herpes is not a vasospastic disease and, consequently, is unlikely to be helped by vasodilatation. To test this supposition, four out of the seven cases were given stellate blocks (Table 10). As expected, this therapy was ineffective and the combined use of corticosteroids did not appear to exert a beneficial influence. Theoretically, cortisone should be helpful by

TABLE 10.—*Herpes Oticus in Seven Cases*

Case	Time Seen, Days	Type of Lesion	Stellate Blocks	Corticosteroids	Recovery	
					Amount	Time
1	11	Complete	3	Nil	None	4 mo.
2	3	Complete	Nil	175 mg. prednisone in 14 days	Complete	21 days
3	4	Incomplete	Nil	Nil	Complete	7 days
4	13	Complete	Nil	175 mg. in 14 days	Incomplete	6 wk.
5	14	Complete	1	Nil	Incomplete	14 mo.
6	11	Complete	1	Nil	Incomplete	6 wk.
7	2	Complete	4	210 mg. in 3 wk.	Incomplete	18 mo.

reducing edematous swelling, but the number of cases is too small to assess its practical usefulness.

Conclusion

This clinical investigation has been an experimental one to determine the treatment most likely to give the greatest degree of recovery in the shortest possible time and to test the validity of the ischemic theory of origin of Bell's palsy. Vasodilatation, produced by paralysis of the cervical sympathetic system by shortening the duration and severity of the paralysis, is shown to be most effective; this finding supports the vasospastic hypothesis. Stellate block is a safe, easily applied method which can be performed in a few minutes in the outpatient department by those possessing the necessary experience and skill.

Active treatment should be begun as early as possible—certainly within the first 24 hours. Usually, the patient with this complaint loses no time in consulting the doctor and certainly looks on the paralysis (which is often thought to be a "stroke") as an "emergency." The attending practitioner should do likewise by considering specific treatment a matter of extreme urgency.

Corticosteroid therapy has not been combined with stellate blocks in most cases in this series because I wished to determine the effectiveness of *one* form of therapy. From a practical point of view, however, the two treatments should be combined: By the one, vasospasm is relieved, and by the other, secondary edema is reduced.

There is still a place for decompression of the facial nerve in the small hard core of cases which fail to respond to this regimen, but this operation will be required less often than formerly. To obtain the maximum benefit from the operation, decompression must be performed before prolonged edema has caused nerve atrophy. It is a mistake to wait too long when there has been no evidence of commencing recovery. When treatment carried out on the above principle fails to show such evidence after a period of four weeks, decompression should be undertaken without delay.

The incidence of Bell's palsy is much greater than an otologist would expect from the number of patients who are ordinarily seen by him, as it is usual for him to be consulted only when there has been nonrecovery after the lapse of several weeks. The active treatment of the early case has been the decisive factor which has directed these patients, in increasing numbers, to me, personally. It is right and proper for the otologist to assume the responsibility for the management throughout the whole course of the condition. The diagnosis demands a full and accurate clinical examination of the auditory system to exclude other peripheral causes of facial paralysis. By virtue of his special training and skill, he is well fitted to perform stellate block, which is the logical treatment for the early case, and by following the case throughout its course, he is in a better position to judge when decompression is needed. For the otologist to carry out his rightful role, it is essential for him to have a clear idea of modern views on the etiology, course, and treatment of this distressing condition. His aim must be to obtain the maximum number of full recoveries in the shortest possible time.

Dr. R. J. Talbot, Director of the Department of Physical Medicine, Hillingdon Hospital, provided some of the "control" cases which were under treatment in his department.

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Oral Antral Fistula Following Zygomatic Fracture Repair

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In recent years, increasing highway speeds have made traumatic injuries of the facial skeleton a commonplace occurrence. Some problems in the management of such injuries, which were not apparent in the past, are now becoming evident. One which we have encountered recently is the development of a postoperative oral-antral fistula through the operative incision following intraoral reduction of fracture of the zygoma. While in no way disabling, this problem may be a source of considerable annoyance to the patient. Also, it may be easily overlooked by the physician who is trying to give the patient the financial benefit of the shortest possible hospitalization.

The so-called "tripod" fracture of the zygoma is one of the more common fractures of the facial skeleton. The injury usually is caused by a hard blow high on the cheek, which separates the heavy part of the zygoma from the adjacent facial bones (Fig. 1). Rowe and Killey's [1] text states:

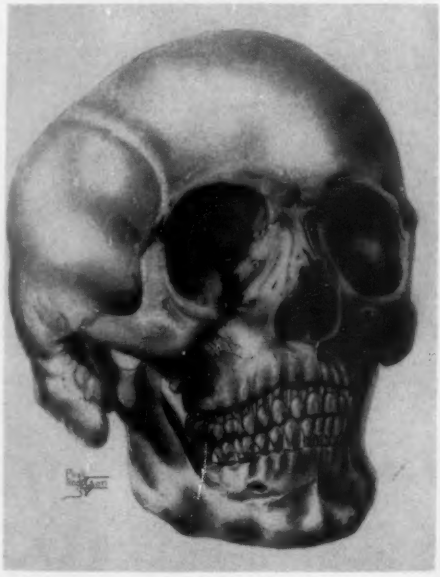
> The most common type of displacement consists essentially of a downward, inward, and posterior rotation of the body of the bone, with a hinge movement and slight separation taking place at the zygomatico-frontal suture, and fracture with little or no displacement through the thinnest part of the zygomatic arch. Separation, usually with an associated step depression, occurs at the junction of the medial third and lateral two-thirds of the inferior orbital rim.

If left uncorrected, the injury usually results in a cosmetic deformity in the form of a flattening of the cheek. It may also produce diplopia by allowing the eye to prolapse into the antrum through a fragmented orbital floor. Trismus often develops, owing to impingement of the depressed zygomatic arch on the coronoid process of the mandible.

One of the simplest approaches to the fracture is through the mouth by an incision in the buccolabial fold. The procedure is described by Kazanjian and Converse [2] as follows:

> Through an incision made in the buccal mucosa above the tuberosity of the maxilla on the affected side, a short, stout elevator is passed upward behind the fractured zygoma. Reduction of the fracture is accomplished by elevating the bone upward and outward; a distinct snap is heard with reduction. . . . In comminuted fracture of the zygoma, additional support is necessary in order to control the position of the fragments. When the floor of the orbit is severely comminuted, the maxillary

Fig. 1.—"Tripod" fracture of zygoma.



Submitted for publication March 21, 1959.

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sinus must be evacuated of detached splinters of bone, the displacement of the zygoma must be corrected, and a means of support provided. An intraoral incision is made through the mucosa over the canine fossa. The anterior wall of the maxillary sinus is exposed, spicules of bone driven into the sinus are removed, the interior of the sinus is exposed, and the hematoma evacuated. A nasoantral opening is made under the inferior turbinate bone to assure adequate drainage. The displaced fragments are manipulated through the intraoral opening in the canine fossa, but will not retain their position unless additional support is provided; for this purpose the cavity of the sinus is packed with gauze through a Penrose-type rubber drain; the drain is removed after a few days through the incision in the mouth or through the nasoantral opening.

Review of a large number of standard texts impresses one with the fact that postoperative sinus infections are rare, that even in the presence of such infections persistence of the surgically created oral-antral fistula is not a significant problem, and that in the absence of prolonged sinus infection a persistent fistula is essentially unheard of. In support of this, a review of the periodical literature for the past 30 years fails to disclose a single reported case of an oral-antral fistula developing after the surgery described. The following reports of two cases treated recently at Walter Reed Army Hospital demonstrate that, while rare, such fistulas can occur and may be as resistant to closure as those seen in the alveolar ridge following tooth extraction.

CASE 1.—A 26-year-old Negro man was brought to the emergency room at Mercy Hospital, Baltimore, on Feb. 10, 1952, after an automobile accident. His injuries were limited to a somewhat comminuted fracture of the right zygoma and fractures of the upper and lower central incisor teeth. After examination, he was transferred to the nearest military medical facility and from there to Walter Reed Army Hospital for definitive therapy. There, on Feb. 14, 1952, an open reduction of the zygomatic fracture was performed via the intraoral approach. A 3 cm. incision was made in the right buccolabial fold from the canine tooth to the last molar. The maxillary antrum was opened widely enough to admit a finger, and the fragments of the fractured zygoma were molded into position with the fingers and an elevator. A nasal antrostomy was made in the inferior meatus, and the antrum was packed with petrolatum gauze passed through the nasoantral window. The intraoral incision was closed completely with interrupted absorbable (chromic gut) surgical sutures.

One-half of the antral pack was removed on Feb. 21 and the remainder on Feb. 27, and the bone retained its position. The nasal antrostomy remained open. On March 11, twenty-six days after surgery, an oral-antral fistula was discovered at the site of the operative incision. A small amount of discharge was present. The sinus was irrigated and the drainage did not recur. The patient was followed for several weeks, during which the sinus remained clean, but the fistula did not close. Finally, on April 16, a Caldwell-Luc procedure was performed. The bone lining the fistula was removed, and a small amount of polypoid tissue in the antrum was curetted free. The nasal antrostomy was widened, and a small pack was passed through it to maintain its patency. The soft tissue edges of the oral-antral fistula were then trimmed and undermined, and the opening was closed with fine chromic gut. Several days later the fistula reopened.

Conservative treatment was decided upon, and on May 16, 19, 23, and 29, the edges of the wound were denuded of epithelium by applications of trichloracetic acid. A small tube was placed in the nasal antrostomy to assure its patency. The fistula remained open.

On June 15, the fistula was sutured closed. The repair promptly broke down.

On June 30, the patient was taken to the operating room, where the fistulous tract was completely excised. A small amount of polypoid mucosa was removed from the antrum, and the nasal antrostomy was widened. No evidence of purulent discharge was found in the antrum. A pedicle flap of buccal mucosa was turned so that the mucosal side faced the antrum. The surrounding buccal mucosa was then undermined and the edges approximated in continuity with the edges of the site of the pedicle flap. This provided a double-layered closure with mucosa facing both the antrum and the oral cavity. The sutures were removed 11 days later, and the repair was seen already to be breaking down. The patient was returned to duty to be followed as an outpatient.

Five months later the patient was readmitted to the hospital. The sinus was clean, the nasal antrostomy was open, and the oral antral fistula was still present. Surgery was again performed. The fistulous tract was excised and a bipedicle flap was turned from the buccal mucosa below the fistulous opening. A pedicle from the buccal mucosa anterior to the fistula was turned so that the mucosa faced the antrum. The adjacent soft tissue was drawn down over the flap and the lower pedicle was closed over the oral surface of the repair, thus providing a three-layered closure with mucosa facing both the antrum and the oral cavity.

Fig. 2 (Case 1).—Fistulous openings in Caldwell-Luc scar, five years after surgery.

Fig. 3 (Case 2).—Fistulous opening in Caldwell-Luc scar, two months after surgery.

The sutures were removed after two weeks. The repair remained intact, and the patient was discharged from the hospital two days later.

Shortly thereafter, he was sent overseas, where close medical observation was not available. He was next examined in the outpatient clinic at Walter Reed Army Hospital in June, 1958. His complaint at that time was unilateral nasal discharge. Questioning elicited the fact that between meals his nose presented no problems, but when eating, and especially when anticipating a meal, if he leaned forward over his plate, clear fluid dripped from his right nostril. Examination showed two fistulous openings in the original operative scar, one almost exactly opposite the orifice of the parotid duct (Fig. 2).

The patient was admitted to the hospital in September, 1958, for elective surgical repair. The fistulous tract was excised and the antrum examined. It was found to be clean except for a small amount of scar tissue, and the nasal antrostomy was wide open. The edges of the fistula were trimmed and undermined, and the wound was closed by an envelope procedure similar to that described by Laskin and Robinson.[8] This type of repair has been used frequently at Walter Reed Army Hospital for fistulas caused by tooth extraction, with good results. After four days, a small opening appeared in the incision. One week later, the opening was still present although the sinus remained clean. The patient was discharged from the hospital to be followed as an outpatient.

Case 2.—A 23-year-old Caucasian man was involved in a fist fight June 21, 1958, and suffered an injury to his right cheek. He was admitted to his local dispensary and transferred to Walter Reed Army Hospital, where x-ray films showed a tripod fracture of the right zygoma. On June 30, an open reduction of the fracture was performed via the intraoral approach. The maxillary antrum was opened, the fragments of bone molded into position with elevator and fingers, a nasal antrostomy created in the inferior meatus, and the antrum packed with iodoform selvage gauze impregnated with Peruvian balsam. The end of the pack was passed through the nasal antrostomy. The intraoral incision was closed completely with 0000 chromic gut.

The antral pack was removed one week later, and the bone remained in good position. The intraoral incision was examined the following day, and a small opening was found extending into the antrum. The sutures were holding well. A very small amount of discharge was present in the fistula, but this cleared within 48 hours without therapy and did not recur. The fistula, however, remained open (Fig. 3).

The edges of the oral-antral fistula were debrided on July 15, and the opening was closed with fine nonabsorbable (silk) surgical sutures. These

Fig. 4.—Opaque dye from antrum outlining oral-antral fistula.

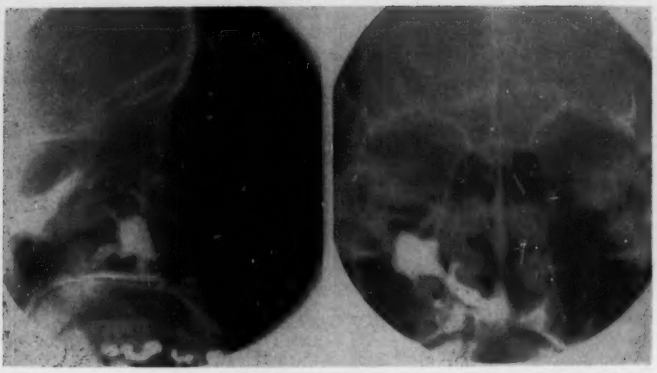

Fig. 5.—Opaque dye from antrum demonstrating patent nasal antrostomy.

were removed one week later, and the fistula remained closed. The nasal antrostomy was open and the sinus was apparently clean.

Two weeks after repair, the patient complained that his wound had reopened, although no perforation could be seen. He was given a soft diet and observed for a few days. Methylene blue was then injected into the sinus through the nasal antrostomy and promptly appeared in the mouth through a tiny opening in the incision scar.

Although small, the fistula did not close spontaneously. In mid-August the patient was taken to the x-ray clinic, where injection of radiopaque dye demonstrated both the oral-antral fistula (Fig. 4) and the wide, patent nasal antrostomy (Fig. 5). The fistulous tract was then excised, the edges of buccal mucosa were undermined, and an envelope closure was made with Dermalon sutures in the manner described by Laskin and Robinson. Four days later, two sutures dropped out and the fistula reopened. The orifice was of pinhole size and, after determining that the sinus was still clean, the patient was returned to duty to be followed as an outpatient. Possible future surgery was contemplated if necessary.

Comment

Both of the cases described showed only isolated zygomatic fractures. Both patients were treated in the same manner, within the optimum time limits for primary reduction. In both cases, the intraoral incision was closed primarily, and both had adequate nasal antrostomies which remained patent. While transient low-grade infections appeared in both patients, these responded easily to therapy and did not recur. In both patients, fistulas developed which could not be closed by either conservative therapy or repeated and, at times, extensive surgical repair. The problem was discussed with the chiefs of the otolarynology,[4] plastic surgery,[5] and oral surgery[6] services, none of whom had previously encountered so resistant a problem. At the time of discharge from the hospital, both patients still maintained open fistulas and were being followed on an out-patient status. Neither showed any sign of sinus infection.

Summary and Conclusions

While repair of fractures of the zygoma via the intraoral approach is ordinarily a relatively simple procedure subject to few complications, it is possible for an oral-antral fistula to develop through the surgical incision which may be extremely resistant to closure. Two cases are presented which demonstrate the occurrence of this complication in spite of precautions taken against it and in the absence of any other difficulty.

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Epithelial Papillomas of the Nasal Cavity and Sinuses

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Epithelial papillomas or polyps of the nasal cavities and paranasal sinuses have been recognized since the first mention by Billroth [1] of a neoplastic growth arranged in a papillary form having an epithelial covering over soft connective tissue. A review of the literature concerning these growths shows confusion as to etiology, pathogenesis, and treatment and is reflected in the variety of names and descriptive terms applied to the lesions, such as epithelial polyps, epithelial papillomas, diffuse or soft nasal papillomas, papilloma durum, and papillary sinusitis.

Epithelial nasal papillomas are uncommon, and although pathologists and clinicians alike have been aware that these growths with their recurrent and malignant potentials are to be distinguished from ordinary nasal polyps, recent references in the literature have been rather sketchy. It is therefore the purpose of this paper to present 10 cases along with a brief discussion of these potentially dangerous lesions.

Methods and Material

At this Veterans Administration Hospital, 10 patients with epithelial papillomas were seen during the period 1947 through 1958. During this period 353 patients with mucous nasal polyps were treated. All polyps are examined, both grossly and microscopically. The ages of these 10 patients ranged from 24 through 61 years. Most of the lesions were located in the nasal cavity, although the sinuses were sometimes involved also. Of these cases, one was lost to follow-up and the remaining nine have been repeatedly examined over periods ranging up to six years. One case has shown recurrences necessitating removal on three occasions, but the lesion has remained benign. Two patients now have malignant lesions which we believe originated in epithelial papillomas. The following case histories are cited.

Submitted for publication March 18, 1959.

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CASE 1.—A 61-year-old white male quadriplegic (due to automobile accident) was first seen for nasal stuffiness in 1952. The symptoms had been present for four years accompanied by a clear watery discharge from the left side of the nose. A nasal polypoid mass was removed by snare at that time. Recurrent polyps were removed in 1955. In 1958, the symptoms recurred and the left nasal cavity was completely occluded with multiple polypoid growths arising from the area of the inferior and middle meatus. Pathological reports on all of the specimens were epithelial papillomas of the nasal cavity.

CASE 2.—This 59-year-old white man was first admitted to a Veterans Administration Hospital in 1952 with inability to breathe through the left side of the nose and pain in the left side of the face behind the left eye. His family physician had noted a nasal polyp and had referred him to this hospital. Examination at that time revealed a grayish, granular, rough mass almost filling the left nasal cavity. It originated beneath the middle turbinate in the region of the ostia of the frontal and maxillary sinuses and measured 2 cm. in length. It bled easily when touched and was removed with a snare. Similar tumor tissue was noted filling the maxillary and ethmoid sinuses when a sinusotomy was done. Pathological report at that time was Schneiderian membrane papilloma with early invasion, and the pathologist added a note stating that he believed the lesion to be an early transitional-cell carcinoma. X-rays at the time revealed pansinusitis with reactive periostitis but no evidence of bone erosion. This patient had recurrences of this lesion five times during the next four years. The tumor was removed each time by surgical excision, and electrocoagulation was utilized to cauterize remaining tissue. He continued to have pain and discharge from the nasal cavity during this period, and several procedures, including avulsion of the infraorbital nerve on the left, were undertaken for relief of pain. In 1956, the patient was first seen in our hospital with recurrent tumor filling the left nasal cavity. A biopsy again showed epithelial papilloma. Further biopsies were interpreted as transitional-cell carcinoma. The patient had excision of tumor from the left nasal cavity, the left maxillary

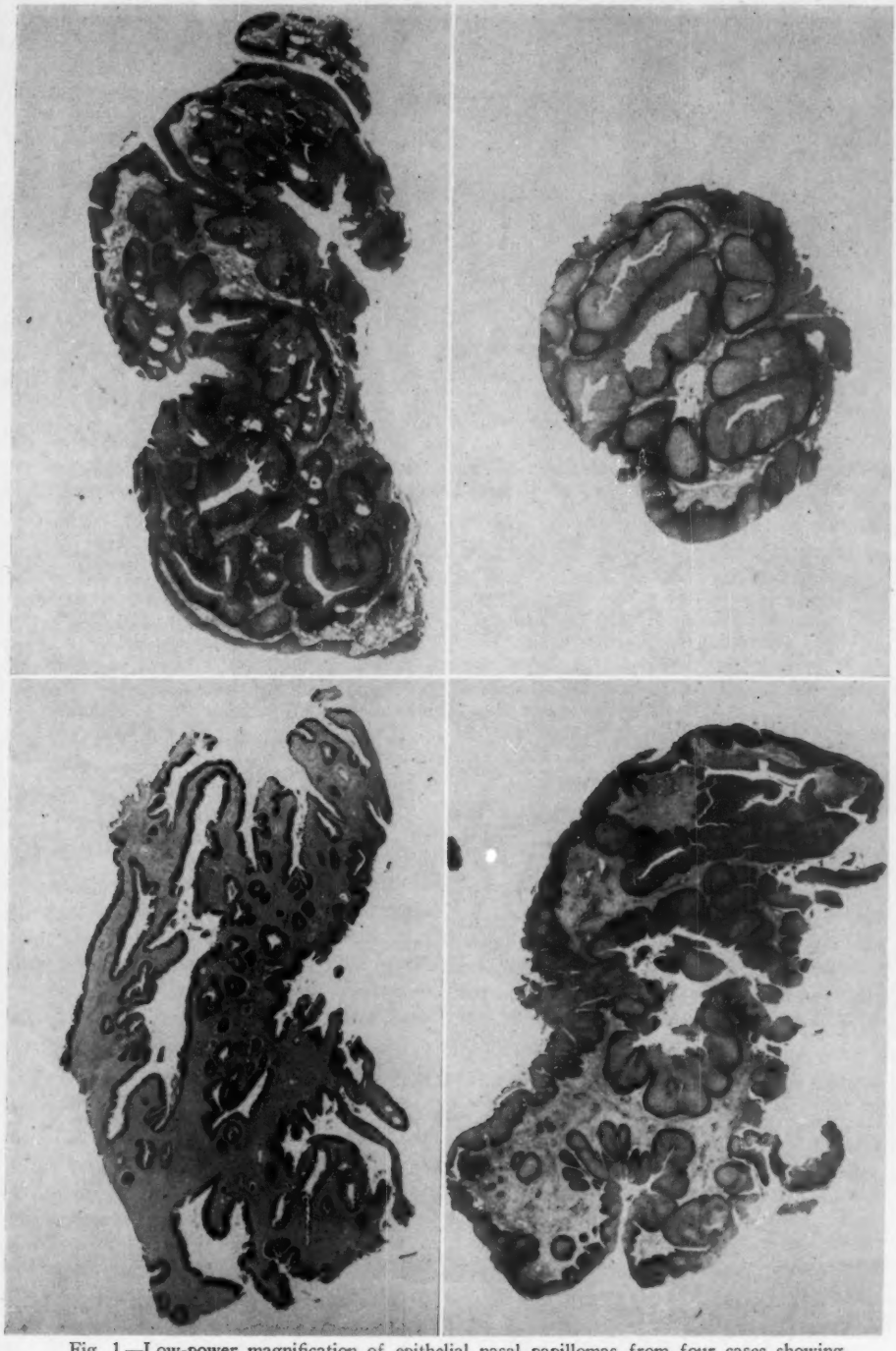

Fig. 1.—Low-power magnification of epithelial nasal papillomas from four cases showing the overgrowth of the epithelial component and the papillary nature of the lesions.

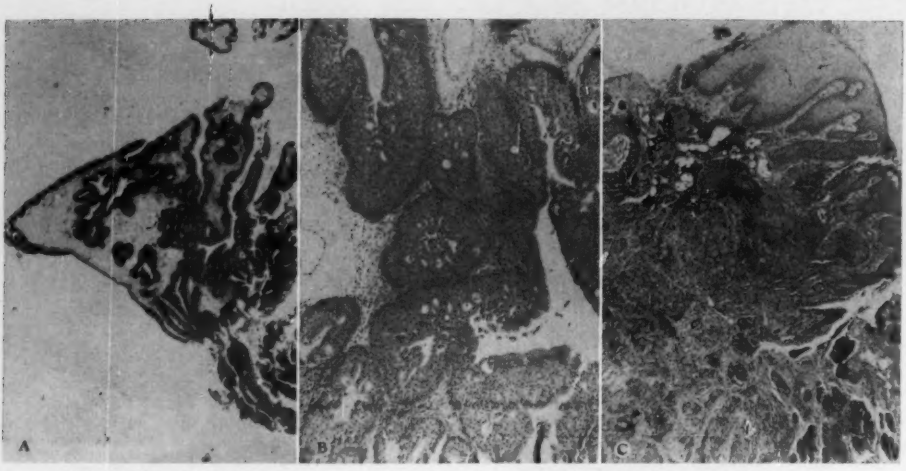

Fig. 2 (Case 2).—*A*, low-power magnification of an early biopsy specimen of nasal papilloma. *B*, medium-power magnification, showing epithelial metaplasia and moderate cellular atypism. *C*, invasive carcinoma in sinus region.

antrum, and the left alveolar ridge. This was followed by x-ray therapy consisting of 6,200 r in 39 days. In addition a small separate squamous-cell carcinoma was removed from the left upper gingiva by excisional biopsy. Several months later the tumor again recurred in the nasal cavity and the left maxillary antrum. Two rubber tubes, each containing 25 mg. of radium, were inserted into the left nasal cavity, giving 2,937 r in 16 days, and one tube containing 25 mg. of radium was placed in the left antral cavity, giving 2,875 r in 64 hours. Four months later the tumor again recurred. Since that time, the tumor has repeatedly recurred and the patient has received palliative therapy.

Case 3.—This 48-year-old white man was admitted in 1957 with proptosis of the right eye, bifrontal headaches, nausea, vomiting, and dizziness. He had had a squamous-cell carcinoma, Grade 1, of the lower lip removed in 1949 and had had basal-cell carcinomas of the nose and inner canthus of the left eye removed in 1953. A right frontal craniotomy had been performed in 1955 and a brain tumor of unknown type treated by radiation therapy. Symptoms had recurred four months prior to admission. X-ray showed a destructive lesion of the right orbit and right frontal bone. After work-up, a right frontotemporal craniotomy was performed and anteriorly and medially a grayish-white tumor mass was found. The wall of the right frontal sinus was eroded and the cavity was filled with tumor. The orbital plate and frontal bone were eroded by tumor and the dura was involved. The tumor in the frontal sinus and that in the brain were very similar and a diagnosis of epidermoid carcinoma was made. A search for a primary was instituted, and a grayish appearance of the right middle turbinate was noted. Pathological report of a biopsy was epithelial papilloma, and again the cells were very similar to the masses of tumor cells noted in the brain. Squamous metaplasia of the lining of the papilloma was present. The patient declined x-ray therapy at that time but has since received x-ray therapy with some improvement.

Comment

Epithelial papillomas are unusual lesions in any given population. Hall [2] collected nine cases between 1918 and 1936. Henriksson [3] reported an incidence of only three newly discovered cases a year in a hospital district of 1,500,000, while Judge,[4] in a discussion of papillary sinusitis, discovered an incidence of 0.8% in routinely examined sinusotomy tissues.

The symptoms manifested by these patients are those commonly found in association with nasal polyps or infection of the paranasal sinuses. Nasal stuffiness and obstruction, postnasal drip, rhinorrhea, and a feeling of fullness over the sinuses frequently associated with headache are the more usual symptoms. The lesions may be asymptomatic when small and may be discovered upon routine examination.

The locations of the lesions vary with the different authors. Kramer and Som[5] found most of the papillomas in their cases occurring in the ethmoid labyrinth, with those of the nasal antrum next commonest. Most of the remaining cases, including our own, have had lesions located in the nasal cavity, although the sinuses were sometimes involved.

Many authors have in the past divided nasal polyps into hard and soft types.[6] Where the epithelial papillomas fit into this description is equivocal. We have found the papillomas in our cases to be small polypoid growths usually projecting from the turbinates in the nasal cavity. They may be multiple or have multiple projections and on closer examination have a granular, grayish appearance with a papillary structure. They vary in size, and the larger ones, which frequently occlude the nasal cavity, are very friable. Bleeding is usually a problem in their removal.

Microscopic examination reveals extensive enfolding of hyperplastic epithelium covering a fibrous stroma which varies in amount. The epithelium is present as nests and projections into the stroma giving the impression of an inverted papillary arrangement. The epithelium varies from stratified squamous to pseudostratified ciliated columnar, but in most cases is transitional in appearance. All variations of epithelium may be seen in the same specimen. The epithelial cells are generally polygonal with oval, granular, basophilic nuclei showing moderate variation in size. The surrounding cytoplasm is eosinophilic near the basilar layers but is frequently clear with distinct borders toward the surface or periphery. Nucleoli are often seen. Mitoses are rather common, particularly toward the basilar layers of cells. Vacuolation is frequent in the epithelial cells and seemingly becomes more apparent with recurrences, but no inclusion bodies or Russell bodies were found in our cases. An accompanying inflammatory reaction is common. Lymphocytes, plasma cells, round cells, polymorphonuclear cells, and eosinophils are found in the fibrous tissue, and microabscesses are occasionally noted in the epithelium.

Some as yet unresolved differences of opinion have existed over whether epithelial papillomas are neoplastic growths or represent responses to inflammatory agents presumably of viral origin. Some recent thoughts on the subject point to an inflammatory origin citing as evidence the presence of inflammatory cells, inclusion bodies and Russell bodies.[7,8] These authors draw attention to the similarities between epithelial papillomas and multiple papillomas of the larynx and venereal warts, although the tendency toward recurrence of nasal papillomas after removal is mentioned.[8] Epithelial papillomas and papillary sinusitis are considered the same process. However, Kramer and Som,[5] in an exhaustive review of 86 cases, stress the pertinent finding that although infection is frequently present, too few epithelial papillomas occur to correlate adequately with the high incidence of nasal inflammation and infection. No particular association with allergy has been noted in previously studied cases.

Most authors draw a distinction between the ordinary mucous nasal polyp and epithelial papillomas, although some, including Ewing,[9] believe that the latter may develop from the former and then may undergo malignant change. Squamous metaplasia is common in the nasal cavity, and small areas are frequently found lining mucous polyps. Kramer and Som[5] discuss the possible evolution of epithelial papillomas from squamous nests or from epithelial heteroplasia or metaplasia.

The malignant potential of these growths has long been suspected, owing to frequent recurrence after removal and more rarely from extension into the cranial cavity by erosion of bone. Seydell[10] suggested that they be regarded as intermediate between benign and malignant neoplastic growths. Hall[2] also felt that they should be regarded as only relatively benign and should be

rechecked frequently. In Willis'[11] book, the malignant counterpart is described as papillary respiratory epithelial carcinoma. Saxen[12] states that he has never seen carcinoma develop from a mucous polyp and if malignancy was associated it was present initially with accompanying inflammation resulting in the polyp. He does, however, draw distinction between nasal polyp and epithelial papilloma.

The conflict as to etiology may not yet be resolved. However, one primary objection in considering the epithelial papilloma inflammatory in origin is the danger of inadequate treatment. We feel that it is safer to consider this entity as a low-grade transitional-cell carcinoma and to treat accordingly with frequent recheck examinations in much the same manner as low grade superficial transitional-cell carcinomas of the urinary bladder are followed.

The treatment of epithelial papillomas is varied and has included electrocoagulation, antibiotics, radiation, and surgery. No treatment has been absolutely satisfactory, and multiple recurrences have been noted. The best results have been in those cases treated by surgical excision followed by radiation and frequent follow-up examinations.

Summary

A discussion of epithelial papillomas of the nasal cavity and paranasal sinuses is presented. Notwithstanding their controversial etiology, the epithelial papillomas are seen as hyperplastic lesions tending toward multiple recurrences and rarely erosion through the bone into the cranial cavity by pressure. The possibility of malignant transformation is cited, and adequate treatment with frequent rechecks is urged.

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Hemangioma of the Jaws

Review of the Literature and Report of a Case

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Hemangiomas of the maxilla and mandible are extremely rare, although they probably occur more frequently than the number of reported cases would indicate. There are both capillary and cavernous types, the cavernous variety having been reported more frequently. This condition is extremely serious in the jaw bones, not only because of the frequent exposure of these bones to trauma but also because their close relation to teeth may necessitate extraction. The simple procedure of tooth extraction or biopsy may have a fatal ending. In several instances the condition was not suspected, and the profuse exsanguinating hemorrhage which followed tooth extraction made the diagnosis obvious. Deaths have been reported from lesions in the maxilla as well as in the mandible and from lesions of both the capillary and the cavernous types. In the majority of reported cases, the lesions have been relatively small, very cellular, and not attended by profuse hemorrhage. Central hemangiomas of the maxilla are more dangerous than those occurring in the mandible; cystic type lesions appear in the maxilla more frequently, are less easily diagnosed, and the blood supply is often more difficult to isolate.

Historical

Bucy and Capp, in 1930,[1] writing on hemangiomas of bone in general, created interest in the lesion by emphasizing its appearance in the roentgenogram. Their article included a complete review of the literature up to 1930, with a tabulation of 28 cases, 5 of which were located in the jaws. Siegmund and Weber, in 1926,[2] reported a small hemangioma of the mandible located near the mental foramen causing fatal hemorrhage following tooth extraction. In 1938, Geschickter and Maseritz[3] reviewed the literature on reported hemangiomas since 1930, as a supplement to that of Bucy and Capp, and added five more cases to the list of hemangiomas occurring in the jaws. Single cases were reported by Brodsky, in 1934,[4] and by Kamphues, in 1936.[5] Since 1938, scattered reports, including the present case report, have appeared in the literature, making a total of 29 hemangiomas of the jaws. Many of these cases were only referred to, while others were more completely reported (Table). Fatal hemorrhage followed tooth extraction in three cases. Severe hemorrhage was not restricted to the large cavernous lesions, for the fatal case recorded by Stoy[6] was that of a small capillary hemangioma of the maxilla. Equally harrowing experiences have been recorded in nonfatal cases. Broderick and Round,[7] in 1933, described an experience as follows:

> Blood spurted for a foot or two from the wound and a most severe arterial bleeding, drenching the clothes and filling the mouth, ensued. I thought that an abnormal palatine blood vessel had been torn—attempts to plug the wound were useless, blood gushing forth as at first, and eventually, after a minute or two, I managed to compress the bleeding point which seemed to be on the edge of the socket, with my finger and stop the bleeding. The child became very pallid and white. . . . No recurrence of the hemorrhage occurred and the plug was allowed to come out gradually; the wound healed well but the patient was white and weak for some time after.

Brodsky (1934)[4] had the following experi-

Submitted for publication March 16, 1959.

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Hemangiomas of the Jaws—Case Reports

No.	Case Report	Sex	Age	Location	Size	Presenting Symptoms	X-Ray Appearance	Treatment	Result
1	Stanley, 1849[5]	M	Child	Mandible	?	?	?	?	?
2	Moltrecht, 1867[6]	M	16	Mandible	?	?	?	?	?
3	Lucke, 1889[6]	F	26	Maxilla	?	?	?	?	?
4	Robin, 1894[6]	?	?	Maxilla	?		?	?	?
5	Pean, 1896[6]	M	50	Maxilla	?	?	?	?	?
6	Siegmund Weber, 1926[8]	?	?	Mandible	Small	?	?	Tooth extraction	Fatal hemorrhage
7	Broderick & Round, 1933[7]	F	11	Maxilla	Large	Hemorrhage around teeth	Ill-defined area radiolucence	Tooth extraction	Sudden & fatal hemorrhage
8	Broderick & Round, 1933[7]	F	12	Maxilla	Large	Swelling, pulsatile pain, hemorrhage	Ill-defined area radiolucence	Extraction, packing, artery ligation	Healed
9	Brodsky, 1934[4]	M	14	Mandible	Large	Bleeding from gingiva	Osteolytic subapical area	Extraction, packing, transfusions, multiple artery ligations	Healed
10	Kamphues, 1936[9]	F	12	Mandible	Small	Bleeding	?	Artery ligation, transfusion, extraction, & packing	Healed
11	Woodward, 1936[9]	M	48	Maxilla Pharynx Larynx	Large	Bleeding	?	Radiation therapy 16,000 mg. hr.	Absence of symptoms
12	Geschickter & Maseritz, 1938[8]	?	?	Nasal cavity Maxilla Base skull	?	?	?	?	?
13	Geschickter & Maseritz, 1938[8]	?	?	Maxilla	?	?	?	?	
14	Watson & McCarthy 1940[10]	?	?	Mandible	?	?	Loculated cavities with fine fibrillary framework	Radiation therapy	?
15	Thomas, 1942[11] Sarcoma Registry	M	?	Mandible	?	?	?	Surgery	?
16	Atlas Dental & Oral Pathology, 1944[11]	F	36	Mandible	Small	None	Routine dental films showed irregularly shaped radiolucent area	Surgery	Healed
17	Stoy, 1947[6]	?	12	Maxilla	Small	Bleeding gingiva	Loss of lamina dura, radiolucent area with displaced teeth	Extraction of teeth	Fatal hemorrhage
18	Erich, 1947[13]	F	55	Mandible	Small	Pain	Multiloculated cystic cavity	Surgery	Healed
19	Sherman, 1951[14]	?	?	Mandible	Large	?	?	?	?
20	Marie & Barety, 1953[15]	F	42	Mandible	Large	Hard swelling	Honeycomb appearance	Radiation therapy 2,700 r, 40 days	Process arrested

21	Marie & Barety, 1953[15]	M	5	Mandible Maxilla	Large	Hard swelling	Honeycomb appearance with erosion of cortex	Mandible surgery followed by recurrence; recurrence treated by radiation, 2,675 r, to jaw; 3 mo. later, 1,100 r; maxilla-radiation therapy	Both lesions showed recession in size & bone regeneration
22	Marie & Barety, 1953[15]	F	23	Mandible	Large	Swelling	Honeycomb appearance with erosion of cortex	2,800 r, 40 days; radiation therapy 6 mo. later, 2,250 r, 33 days	Regrowth of bone; process arrested
23	Pizer, 1954[16]	F	32	Mandible	Small	None	Ill-defined area of radiolucence	Surgery	Healed
24	Williams & Gilbert, 1955[17]	M	11	Mandible	Small	Swelling and attacks of throbbing	Cyst-like areas of rarefaction	Surgery	Healed
25	Villa & Limchayseng, 1955[18]	F	18	Mandible	Small	Hard swelling	Honeycomb appearance	Surgery	Healed
26	Battersby, 1957[19]	M	15	Mandible	Large	None	Extensive osteolytic changes	Surgery	Healed
27	Kilgour, 1957[20]	F	40	Mandible	Large	Swelling	Fine soap bubbles & radiating trabeculations	Block dissection of area, later	Healed
28	Kilgour, 1957[20]	M	?	Mandible	Small	None	Few loculations in a cystic area	Surgery	Healed
29	Present case	F	25	Mandible	Large	Bleeding, swelling, pulsating pressure	Large multicystic cavities showing honeycomb appearance with erosion of cortex	Biopsy followed by radiation, 2,464 r, 29 days	Process arrested; regrowth of bone

ence with a mandibular cavernous hemangioma:

The molar was extracted; it "popped" out of the socket, after which there was a profusion of blood spurting with terrific velocity from the mouth. The hemorrhage was stopped with great difficulty. The disease, however, progressed and in about a year the premolars and the first molar teeth were loose and slightly painful. X-ray examination showed practically the entire right side of the mandible involved. The treatment consisted of ligation of the right external carotid artery which did not prevent extreme hemorrhage at the operation performed later. By means of compression with a metal splint and transfusions, the hemorrhage was arrested. After ligation of the right common carotid artery and the left external carotid, the splint was removed, which again was followed by prodigious hemorrhage. The final treatment consisted of trephining the bone and inserting the electrocautery immediately into the hole and then injecting into the tumor sterile bone wax mixed with lipiodol. This resulted in an occlusion of the blood sinuses and regeneration of bone.

Clinical Findings

Hemangiomas of the jaws may present no clinical symptoms when small in size or in the early stage of expansion. The sex and age of the patient seem unrelated. In 18 cases the presenting symptoms are recorded. Mucosal bleeding is present in six cases; swelling, in eight cases; pain, in one case, and a throbbing pressure sensation occurred in three. In 3 cases no symptoms are present, and in 11 other cases the symptoms are not known. In two instances, Brodsky [4] and the present case report, the early presenting symptoms are dependent upon the presence of teeth. Bleeding from the mucosal surface around the teeth occurs long before any large area in the bone is defined. This mucosal bleeding is of a peculiar type in contrast to most gingival bleeding in that it is spurting in character instead of an indolent ooze. This gingival bleeding stops upon extraction of the teeth, and when the bone lesion suddenly begins to enlarge different presenting symptoms occur.

Roentgen Findings

Perhaps the roentgen findings are the most helpful in obtaining a working clinical diagnosis. This is especially true of the larger lesions in the mandible undergoing active proliferation. The picture is one of multicystic areas of bone resorption, producing a soap-bubble or honeycomb effect. The areas of resorption are irregular in outline and have indefinite borders which blend into the normal bone architecture. Fine trabeculations are present in some, whereas large cavities are present in others. Erosion of the lamina dura, with resorption and displacement of the dental roots, has been observed. The cortices of the bone may become thinned, expanded, and, in some instances, eroded through. Geschickter and Maseritz [3] observed several small punched-out areas, distal to the lower border of the tumor, and state that these areas are important because they indicate progression and are typical of angioma. The above findings have been helpful in lesions of the mandible but not particularly helpful in the maxilla. The nasal and paranasal sinus cavities, plus superimposed cranial structures, make these changes less distinctive in the upper jaw roentgenograms. Unfortunately, the small, equally hazardous, early lesion has no distinguishing characteristics in the roentgenograms.

Pathology

The American College of Surgeons classifies angiomas of bone as a variety of tumors of nonosteogenic origin. Angiomas involving bone are of two types; those arising from blood vessels within the bone and affecting bone primarily, and those arising from blood vessels of the periosteum and affecting bone secondarily. These blood vessel tumors have an embryonic capacity for irregular and unrestrained growth. According to Watson and McCarthy [10]:

The average hemangioma pursues a benign course and if untreated grows slowly until adult life. Occasionally one will suddenly develop rapid acceleration of growth, become locally destructive, and may even cause death by hemorrhage or sepsis if not treated promptly. These lesions enlarge and develop by extending solid "buds" of endothelium into adjacent bone. The solid cords

of endothelial tissue then become canalized and establish communication with the parent vessel. Watson and McCarthy[10] also felt that many which did not appear until adult life were present much earlier but were not noticed until they suddenly began to enlarge. The head and neck area has a high percentage of the total number of hemangiomas of bone, and there is some tendency for the occurrence of more than one lesion in the same patient. Since hemangiomas of bone are usually a histologically benign lesion, the type (whether capillary or cavernous) is mostly academic. By the time the tissue reaches the laboratory, the fate of the patient is usually decided. The cavernous lesions usually do not provide an opportunity to obtain tissue. Those lesions from which tissue can be obtained require histologic evaluation. Often, there is no unusual bleeding at surgery to indicate the vascular nature of the lesion, and its true nature is disclosed only through histologic study.

Report of Case

A 25-year-old mother of three children presented with a swelling of the left side of her face, numbness of the lower lip, and a sense of pressure of 12 months' duration in the left mandible.

Eight years prior to admission the patient had her lower left wisdom tooth removed because of its impacted position. The surgery was reportedly attended by marked hemorrhage, and there was trouble with hemorrhage in the postoperative period.

Four years prior to admission, the patient had her lower left molars and bicuspids removed because of a marked "condition of pyorrhea" with loss of alveolar bone and continuous gingival bleeding, at times spurting in character. All other teeth were present and in excellent condition, with normal alveolar bone and gingival tissue. Moderate to marked bleeding was encountered with these extractions but was readily controlled. Six months later, the alveolar bone was exposed, and an attempt was made to create enough space to construct a lower partial denture for replacement of the extracted teeth. This procedure was terminated short of providing adequate space because of marked bleeding.

One year prior to admission, the patient gradually became aware of a sense of pressure at the angle of the mandible associated with intermittent numbness of the left side of the lower lip. The sense of pressure and the numbness gradually increased in intensity until the time of admission.

Two months before admission, intraoral dental films revealed a peculiar porosity of the alveolar bone near the mental foramen. Conventional posteroanterior and lateral jaw films (Fig. 1) showed an ill-defined multilocular cyst which extended throughout the whole left jaw up to the mandibular notch. The cortex was expanded and thin, with the mandibular canal outline obliterated. Several small punched-out areas were observed in the distal portion of the lesion, creating a soap-bubble or honeycomb effect. The patient was referred to me for further diagnostic procedures.

Admission physical examination was essentially normal except for findings related to the left portion of the mandible. The ascending ramus and angle of the mandible were prominent, with

Fig. 1.—Roentgenograms of mandible before treatment, showing honeycomb effect and cystic area.

Fig. 2.—Photograph of the mouth, showing expansion of the left inferior alveolar process and swelling of left cheek.

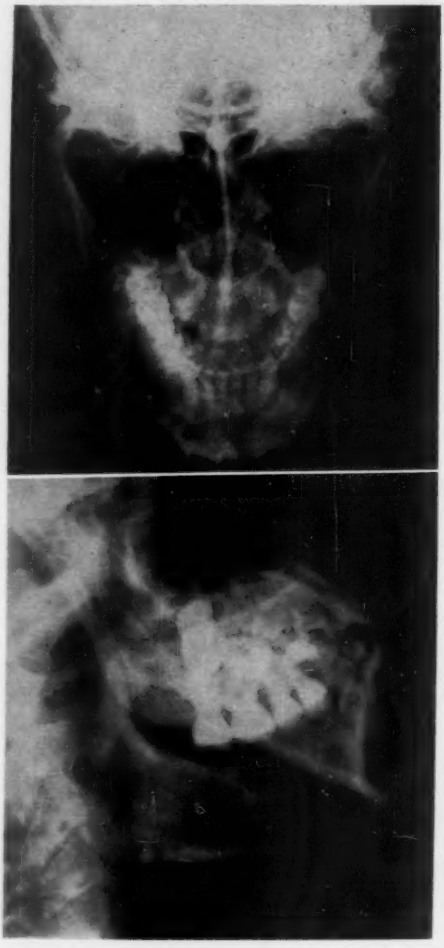
a generalized fullness extending forward over the posterior portion of the horizontal ramus. The alveolar ridge was raised and widened in the area of missing teeth (Fig. 2). The veins in the mucobuccal fold at the angle of the jaw were slightly prominent. With the teeth in centric occlusion, the space between the lower alveolar ridge and the opposing maxillary teeth was almost obliterated. The mandible moved freely in all directions without pain. There was a definite zone of paresthesia corresponding to the skin distribution of the left mental nerve.

In addition to routine admission blood and urine studies, the following laboratory procedures were performed: serology, bleeding time, clotting time, NPN, FBS, total protein with A/G ratio, prothrombin time, calcium, phosphorus, liver-function tests, transaminase, and alkaline phosphatase. The above studies were all within normal limits. The first-strength PPD was negative and the urine found to be free of Bence Jones protein.

The x-ray metastatic series was negative. Laminagrams of the jaw were taken and confirmed the findings reported from earlier conventional x-rays.

On the third hospital day, a biopsy of the area was performed under block anesthesia, with malignant tumor of the mandible as a working diagnosis. The patient was placed in a semi-Fowler position, and a linear incision 2 cm. long was made over the midline of the alveolar ridge, extending forward from the beginning of the ascending ramus. The mucoperiosteum was elevated over both the lingual and buccal surfaces, without extension of the incision to provide flaps. The periosteum was found to be very adherent and was carefully elevated, exposing the alveolar bone cortex which had a porous appearance. Blood came forth briskly from the small holes and was nearly spurting in character. This bleeding was readily controlled with gauze pressure. In view of the highly vascular nature of the bone, plans to chisel out a section of the alveolus were changed in favor of creating a small fenestra with a bone burr. Fracture of the thin cortex of bone was thus avoided and the size of the bone opening limited should pressure be required to control the bleeding. An oval section of the bone about 4 mm. in diameter was removed without difficulty or excess bleeding. A bulging grayish-blue tissue was seen through the bone opening. This tissue was touched with a blunt probe to determine the consistency of the soft tissue mass, when suddenly the bulging membrane became a bubbling fountain of blood which, in a matter of seconds, filled the mouth and flowed over the patient onto the floor. During the seconds required to drop

Fig. 3.—Roentgenograms of mandible eight months after therapy, showing arrest of lesion with increased density of bone and loss of honeycomb effect.



the probing instrument, grasp the mandible with the right hand and move the left thumb over the fountain of blood, the patient became unconscious and chalky white in color. The flow of blood, although easily controlled with thumb pressure, would commence again as vigorously as before once the pressure was released. Consciousness and normal skin color returned as soon as the flow of blood was staunched, and the patient was placed in a horizontal position. Absorbable gelatin sponge (Gelfoam), crushed into firm balls 6 mm. in diameter, was forced into the bone cavity under the left thumb. When a number of these absorbable gelatin sponge pieces had been inserted, a ball-valve effect was obtained and the hemorrhage controlled. During the time of packing, an instrument was passed at least two inches into the cavity without contacting any soft tissue. The mucoperiosteum was closed tightly with interrupted 000 absorbable (chromic gut) surgical sutures. The total estimated blood loss was 750 cc. The patient regained consciousness readily but remained confused for several hours, even though the vital signs were stable. The biopsy area healed without incident of hemorrhage, the lesion continued to expand, and the previous symptoms became more intense prior to starting radiation therapy.

Radiation therapy was started approximately one month after the biopsy procedure. A total dose of 2,464 r was delivered to the jaw over a period of 29 days.

Eight months after completion of the radiation therapy, the facial asymmetry had disappeared. Symptoms of pressure in the jaw and lip numbness had markedly decreased. X-ray examination at that time revealed a filling in of the active zone of destruction with new bone. There was a decrease in size of the larger bone cavities and increased density of the surrounding bone with visualization of the mandibular canal (Fig. 3).

Comment

Hemangiomas of the jaws seem to be commoner than previous case reports have indicated. They have not been of sufficient numbers, however, to receive routine differential diagnostic consideration in cystic lesions of the jaws. Routine approach to cystic lesions of the maxilla and mandible should be modified to provide safety for the patient should this diagnosis become obvious during a surgical procedure of excision or diagnostic biopsy. The experiences recorded in this paper should make one aware that this is a dangerous lesion and can result in death for the patient in a matter of moments, regardless of the size of the lesion or its location.

The maxillary hemangioma is a more difficult and dangerous lesion, not only for diagnosis but for control of profuse hemorrhage should such be encountered. The blood supply is more likely to be from multiple sources, and ligation of the major arteries may not influence the flow of blood appreciably. Lesions in the maxilla may expand into the air-containing nasal and paranasal spaces and become very large. The frequent occurrence of multiple developmental cysts in the maxillary area tends to divert differential diagnostic consideration away from vascular lesions.

Hemangiomas of the mandible lend themselves to more accurate evaluation. The lesion in the mandible may take on the characteristics of hemangioma of the long bones. X-ray examination can isolate the mandible very well from other facial and cerebral structures. The blood supply is more definite, and in large lesions the periosteal components are more easily visualized.

Regardless of the history, clinical examination, and x-ray appearance of the lesion, one must have a more definite working diagnosis before final treatment is undertaken. With hemangioma of the bone as a consideration in the working diagnosis, one can modify the approach so that a major hemorrhage, when encountered, will not be a fatal one. The incision should be a straight line so it can be easily and effectively closed. It should be in an area where direct force can be most easily applied and maintained if a pressure dressing becomes necessary. If the lesion is found to be neoplastic instead of vascular, a limited incision will be well within the confines of a future block excision without unnecessary sacrifice of adjacent normal facial tissue. The periosteum should be elevated with care so that the overlying bone or a small part of the lesion can be thoroughly inspected. This is particularly true in the

area showing a honeycomb appearance on the x-ray, for there are small vessels leading from the marrow spaces through the cortex to the periosteum. These vessels will spurt on severance but gradually cease bleeding with applied pressure. The flow of blood increases as the more superficial aspect of the lesion is approached. Large bone flaps are to be avoided until the contents or nature of the lesion is better known. Chisels should be avoided where the cortex is thin and a wide break-through likely to occur. The overlying bone may be carefully fenestrated with a burr so that the lining can be observed, and the contents of the space may be evaluated by probing or needle aspiration. Care in the diagnostic biopsy may avoid the need for heroic surgical procedures undertaken under adverse circumstances and often without adequate help, facilities, or anesthesia.

Treatment of these lesions has been recorded in a number of cases. Large actively expanding lesions of both the maxilla and mandible have been successfully treated by heroic surgery. Deaths have been recorded from lesions both large and small as the result of surgical interference. The greatest danger to the patient is not from any potentially malignant aspect of the lesion but from the very great danger of fatal hemorrhage. These lesions, when rapidly expanding, have to be treated in some manner to avoid a spontaneous fatal hemorrhage.

Large lesions in both the maxilla and mandible have been treated by radiation therapy. Radiation therapy has been successful in large lesions even where the cortex has been eroded, both as a means of primary treatment and where primary surgical intervention was followed by recurrence of the lesion. Radiation therapy would seem to be the procedure of choice in lesions not in dire need of hemorrhage control. Unfortunately, as is often the case, tooth extraction precipitates both the diagnosis and a dire need for hemorrhage control.

Should surgery be required for a large lesion, appropriate vessel ligation, preparation for rapid and numerous transfusions, and immediate packing of the cavity must be considered. Vessel ligations reported by Broderick and Round [7] and Brodsky [4] seem not to have influenced the flow of blood, and, with this in mind, one should concentrate on controlling the immediate hemorrhage by obliterating the cavity with absorbable packing material.

Summary

Twenty-nine reported cases of hemangioma of the jaws are tabulated. The diagnostic and therapeutic problems associated with this type of lesion are presented.

Hemangiomas should be considered more frequently in the differential diagnosis of radiolucent lesions of the jaws because of the very great danger of unexpected fatal hemorrhage.

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The Clinical Use of T.O.E. in E.E.N.T. Practice

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Introduction

My interest in this subject dates from my military service in the Navy during World War II, at which time all of the medical officers were plagued with patients with external fungous infections of the skin and especially the external ear. I recall, on my first day of duty at the Naval Air Station on the Gulf of Mexico in Texas, when I was presented with 52 cases of otomycosis.

Definition.—T.O.E. refers to the combined extract of Trichophytin, Oidiomycetes, and Epidermophyton, which represent the three important groups of pathogenic fungi known to produce allergic symptoms.

History.—Mycotic infections are noted for three characteristics: (1) their resistance to treatment, (2) their recurrence, and (3) their complications. Recent research has indicated that the resistance to treatment as well as the recurrences are due to altered sensitivity.[1]

This paper is concerned with the allergic complications of the skin and mucous membrane (or the dermatophytid). The "id" reaction (or dermatophytid) is known to be precipitated by the recurrence or chronicity of mycotic infections, irritative treatment such as x-ray, strong chemicals or drugs (notably benzoic and salicylic acid [Whitfield's] ointment), and pyogenic involvement of the open lesions or excoriations in the epidermis as well as the mucous membrane.

Submitted for publication March 2, 1959.

Presented at the Houston Meeting of the Gulf Coast Allergy Study Group, on March 2, 1957, and at Akron, Ohio, Academy of Ophthalmology and Otorhinolaryngology, on Dec. 7, 1957.

Diagnosis

Clinical Symptoms.—The diagnosis of the "dermatophytid" reaction is dependent upon the absence of the so-called mycotic pathogens in the lesions, changes in the skin or membranes such as the vesicular eruptions, the so-called "dry excoriations" or cracks in the skin or the mucous membranes, the swelling and erythema of the areas involved, and, most important of all, the varying degrees of pruritus, from mild to intolerable, regardless of the area involved. Involvement of the mucous membrane may be not only erythematous and discolored but also may be characterized by the so-called "black" or "hairy tongue" which varies from brown to black color and also the so-called "monilial" type of pharyngitis which is characterized by a white tongue from which no pathogens can be obtained. In addition to these, involvement of the gastrointestinal tract is very often characterized by dyspepsia, aerophagia, flatulence, and chronic heart burn.

Symptom Distribution.—Two hundred seventy patients were studied in this series. Of these, 3 presented symptoms of headaches, 5 had hives associated with skin lesions, 5 had a cough as their presenting symptoms, 5 had scalp lesions, 12 had gastrointestinal symptoms, 12 had a rash of the eyelids, 23 had mouth symptoms, 35 had lesions of their fingers only, 59 had ear symptoms such as the otomycosis syndrome with the "id" reactions, and 111 had skin lesions other than their fingers. The number of patients studied since the March 2 presentation is 602.

Treatment

History.—Originally, the use of allergenic extract in the treatment of mycotic or allergic complications of mycotic infections fell into disrepute

from the beginning, since it was found that the patients' symptoms often became aggravated from this type of treatment. This paper is concerned primarily with this fact, since the use of the skin titration method first reported by Rinkel[8] is particularly suited to this type of treatment for four reasons.

First, there is the safety factor involved which allows one to treat these patients without danger of precipitating increased symptoms.

Second, it is used to confirm the diagnosis, since the reaction to stronger solutions would be missed for fear of exacerbation. This second reason is obvious when one considers the fact that most conventional systems of today and most of the methods of allergy in the past have dealt with only one or two different strength solutions. In the use of the conventional or older method, if one treated with a solution any weaker than 1 :100, he would miss entirely those that react on 1:100. The group who react on stronger solutions represent the largest group of those that we tested. Those who test with a solution of only 1:1,000, 1:10,000, or 1 :200,000 would miss the great majority of patients with such a system (as will be shown later in the paper).

Third, the degree of sensitivity changes rapidly in these patients and, therefore, the first testing might result in failure if one depended entirely on this test.

Fourth, the titration would also prevent failure in regard to concomitant food factors,[3] which also change the degree of reaction, since the patient would become less sensitive with their omission as a rule and, therefore, the solution would be too weak to give them proper relief.

Titration.—These patients are titrated as coseasonal patients according to the method of Rinkel,[8] and it is often well to use the No. 12 dilution as a starting point for titration if extreme sensitivity is suspected. Of these patients tested for titration, 190 reacted on No. 1 solution, 32 reacted on No. 2 solution, 25 reacted on No. 3 solution, 19 reacted on No. 4 solution, 3 reacted on No. 5 solution, and 1 reacted on No. 9 solution on original testing. At this point, however, it should be emphasized that even with this one patient, titration should be started beyond the point of sensitivity to avoid exacerbation of the symptoms. One should start with at least No. 9 solution for titration in all patients. In titrating these by coseasonal technique, the end-point dilution usually produces a 6 or 7 mm. wheal, but with a distinct zone of erythema. This differs from the penicillin end-point, since penicillin will create an erythematous papule and does not have its own erythema around it. When a 6 mm. erythematous wheal is obtained as an end-point, then the fourth dilution (including the 6 mm. wheal) or three dilutions weaker than the end-point is used as the solution to start treatment with. For instance, if an end-point is reached on No. 6 solution, then treatment would be started with the No. 9 solution. Table 1 illustrates this point.

TABLE 1.—*Use of Various Dilutions of T.O.E.*

1:39,062,500	9	5
1:7,812,500	8	5
1:7,562,500	7	5
1:312,500	6	6
1:62,500	5	
1:12,500	4	
1:2,500	3	
1:500	2	
1:100	1	
1:20	0 Concentrate	

These patients are treated with a 10× dose of the fourth dilution weaker than the end-point; then, if no improvement is obtained, they are treated by advancing doses, increasing the treatment to the stronger dilution or until a clinical response is obtained. It should be noted, however, that if, on the original testing (since the end-point is considered to be fractional), the patients show improvement in their symptoms, one can proceed by giving double this amount or perhaps increasing it by 50%. When the patient reacts with an exacerbation of symptoms of the original application of testing, one must go to the fourth dilution, including the end-point, and start treatment with this weaker dilution and proceed to treat them with an increasing dose until an optimum dose is obtained. The interval between treatments will depend upon each person; in fact, the beginning doses may be given at three-day intervals as a matter of convenience to the patient. If an interval can be established by treatment which gives relief, then no succeeding treatment should be given as long as relief is obtained. In other words, when a patient obtains relief, the next treatment should be given at the return of symptoms. If a definite interval when the symptoms return can be found, one may precede this interval by 24 hours and give the dose prophylactically to prevent the return of symptoms.

Changes in Sensitivity.—Three factors which may alter the sensitivity or cause a change in sensitivity are, first, treatment itself; second, inhalant allergens; and third, concomitant food sensitivity. These factors must all be considered in treatment.

Changes in Sensitivity Due to Treatment.—After continued treatment, it may be noted that there is no longer clinical response and, therefore, one should consider the possibility of a shifting end-point either to the right or left. The patient should be retested for the establishment of a new end-point. This factor is the result of treatment it-

self, and the majority of patients shift to the right after treatment, as they do in other treatments with titration methods.

Inhalant Factors.—The concomitant reaction of these patients to molds and house dust has also been established,[8] and the majority of these patients also react to airborne mold spores, house dust, and plant pollens. It is well to establish the diagnosis immediately when the titration is done—in other words, to titrate the patient for inhalant factors

TABLE 2

YEAST: A BRIEF DESCRIPTION OF COMMON SOURCES OF CONTACTS

Compiled by Dor W. Brown, Jr. M. D.

1. The following foods contain yeast as an additive ingredient in preparation (often called leavening)

Breads Hamburger Buns Rolls, homemade or canned Cookies
Crackers Hot dog Buns Canned Ice Box Biscuits: Borden, Pillsbury and
Pastries Cake & Cake Mix General Mills.
Pretzels Flour enriched with vitamins from yeast: General Mills, Inc., Flour Corporation flour and enrichment wafers, Pfizer Laboratories enrichment products.
Milk fortified with vitamins from yeast Meat fried in cracker crumbs

2. The following substances contain yeast or yeast like substances, because of their nature or nature of their manufacture or preparation.

Mushrooms Truffles Cheeses of all kinds including Cottage Cheese Buttermilk
Vinegars: Apple, Pear, Grape and distilled. These may be used as such or they will used in these foods,—

Catsup Mayonnaise Olives Pickles Sauerkraut Condiments Horse radish French dressing
Salad dressing Bar B—Q sauce Tomato sauce Chili peppers et cetera
Mince pie Gerber's Oatmeal & Barley Cereal

Fermented Beverages: Whiskey, Wine, Brandy, Gin, Rum, Vodka, Root beer et cetera
Malted products: Cereals, Candy and milk drinks which have been malted.
Citrus fruit juices, either frozen or canned. Only home squeezed are yeast free!
Dermison rest capsules

3. The following contain substances that are derived from yeast or have their source from yeast.

Vitamin "B" capsules or tablets *if made from yeast*
Multiple vitamins, capsules or tablets with Vitamin "B" made from yeast.
Zylax Zymelose Zymenol Lilly's vitamin products which contain B—12
U. S. Vitamin products: Laxo—Funk, Phoscaron—D, Vi—Litron Drops,
Mead Johnson's Vitamins which contain B—12
Squibb's Vitamin products if indicated on the label.
Parke Davis's Vitamin products: VIBEX
Merck, Sharp & Dohme Vitamin products which contain B—12
Lederle's Vitamin products. Endo's Vitamin products: Manibee tablets, S. C. T.
Massengill Vitamins.

4. The following products are known to be free of yeast as of Dec., 1, 1957

Pioneer Flour Mills Gladiola Flour Mills International Flour Mills
Abbot's Vitamins: Dayalets, Vidaylin et cetera
Robin's Vitamins: Allhee with C.
U. S. Vitamin products except: Laxo—Funk, Phoscaron—D and Vi-Litron
Mead Johnson's Vitamins if free of B—12
Squibb's Vitamins if not indicated on the label.
Parke Davis Vitamins except VIBEX
Merck Sharp & Dohme's Vitamins except those containing B-12
Upjohn's Vitamin products Schenleys Vitamin products
Hoffmann-La Roche's Vitamin products
Endo's Vitamin products except Manibee Tablets, S. C. T.
Flour enrichment wafers or products of Merck and Hoffmann-La Roche

5. INSTRUCTIONS:

if this has not been done previously, and it will be found that the patients will respond more satisfactorily if they are treated with all of the inhalant substances to which they are sensitive, especially in the case of the mold-sensitive patients. Some of the airborne molds are titrated in groups, and, in our locality, we titrate for the following molds as a group: Aspergillus niger, Botrytis cinereus, Chaetomium globosum sp., Fusarium moniliforme, Helminthosporium sp., Monotpspora lanuginosa, Mucor heimalis, Penicillium atramentosum, Phoma destructiva, Phizopus arrhizus, and Phodotorula sp. We also titrate for the groups of smuts, such as the following: Johnson grass, Bermuda grass, wheat, corn, oat, barley, sorghum, milo, maize, and foxtail. Alternaris, Hormodendrum, and Cephalsporium are always titrated individually. Seasonal changes in pollens, house dust, and molds may affect response to treatment and should be anticipated.

Concomitant Food Factors.—Symptoms from yeast-containing foods are so frequent in these patients that this concomitant factor must be considered in all patients. *Yeast-containing foods must be eliminated completely from the diet in order to relieve the symptoms of patients with concomitant yeast sensitization.* It should be remembered that when these yeast-containing foods are eliminated from the diet, the end-point of the reaction will frequently be altered, usually by a shifting to the right, and therefore, these patients must be retitrated in order to establish their new end-point if they fail to respond. The yeast-containing foods are listed in Table 2.

Local Treatment.—Mycotic reactions in our hands have been notably resistant to any type of local treatment except a very conservative type. Soaks with diluted 1:5,000 potassium permanganate solution, as well as aluminum acetate (Burow's) solution, have been known to benefit some of the patients temporarily and yet later to aggravate their symptoms, owing to subsequent cracking and drying of the skin. Permanent relief has been delayed as well as prevented by this type of treatment. It has also been our experience that local use of antibiotics and local use of cortisone and cortisone derivatives or compounds very often aggravate these conditions rather than relieve them as has been reported in the literature. The majority of these patients clear up without local treatment and benefit from local treatment only temporarily until the condition is permanently relieved. Of the many preparations that we have tried, only one preparation has proved beneficial in our hands as far as local treatment is concerned, and that is a topical ointment of 3% oxytetracycline hydrochloride and 1% hydrocortisone combined in a petrolatum base.* The ingredients of this ointment have been tried separately, with aggravation of symptoms. Local treatment with this alone without titration is ineffective.

Prophylaxis.—It has been observed in these patients that prolonged exposure to dampness and damp weather will precipitate symptoms, especially in patients with involvement of the ears. The ear patients, especially, notice this from swimming, washing their hair, and excessive perspiration in the summer, when moisture is in the ear canal for a long period of time. It is well to advise these patients against prolonged exposure to moisture and to dry the exposed surfaces of the skin as soon as possible.

The use of antibiotics, especially the broad spectrum group, orally, often precipitates symptoms in these patients and should be avoided if at all possible, except in cases of dire emergency. If antibiotics are used, then the so-called "mycotic" inhititors should be given simultaneously with the antibiotic and, if at all possible, the oral use of antibiotics should be avoided in these patients. The parenteral use of antibiotics is to be preferred.

Summary

The definition and history of T.O.E. is given. The diagnosis and distribution of the "id" reaction is discussed. Treatment of these patients by T.O.E. titration is outlined, and the other factors to be considered are included as variables. Local treatment and prophylaxis are discussed.

Conclusions

1. Treatment of "id" reactions in the past has been unsatisfactory.

2. Titration with T.O.E. is a safe and satisfactory method of treatment for "id" reactions.

3. Variable factors other than T.O.E. affect the titration treatment and must be considered.

4. Local treatment of "id" reactions alone is not effective and in some cases harmful.

5. Prophylaxis of "id"-producing factors should be considered in these patients.

6. Because of the work of R. L. Baer et al., as well as the work which we have

* Terracortril, made by Pfizer Laboratories, 630 Flushing Ave., Brooklyn 6.

done, the interesting possibility of treating active fungus infections with such a method presents itself.

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Eosinophilic Granuloma of the Temporal Bone

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The occurrence of a case of eosinophilic granuloma in the temporal bone of a child has interested me, and caused me to survey the literature and study the present concepts of this rare disorder.

Eosinophilic granuloma is a locally destructive lesion. When it involves the temporal bone it is usually associated with pain and serosanguinous discharge from the ear. It is characterized, clinically, by a soft and friable granulation tissue occluding the external auditory meatus and, radiologically, by punched-out areas in the bone. Reports on such lesions by Schuknecht and Perlman,[9] Dingley,[4] and Dawes[3] have established the disease as a distinct entity.

Pathology

Microscopically, the involved area contains a friable brownish-white granulation tissue. The outstanding feature is an occurrence of a large number of histiocytes, and so the lesion has the appearance of a granuloma; this, together with the large number of eosinophils, has led to the use of the term eosinophilic granuloma.

Lymphocytes and plasma cells are seen frequently, and areas of necrosis and hemorrhages may be found at places. The resulting cholesterol crystals are commonly seen surrounded by foreign-body giant cells (Fig. 1).

As the lesion progresses, the histiocytes pick up lipid and show a foamy appearance. Later, these lipophages dominate the field, and the eosinophils tend to disappear. This phase is well described by the term lipogranuloma. In the final stage, fibrosis, and even ossification, may be seen. Thus, it is a self-limiting lesion with a tendency to heal spontaneously, but should the granuloma reach the surface, secondary infection supervenes.

The basic character of the lesion is similar to that of the bony lesions in Letterer-Siwe disease and Hand-Schüller-Christian disease, and so these disorders are grouped

Submitted for publication March 26, 1959.

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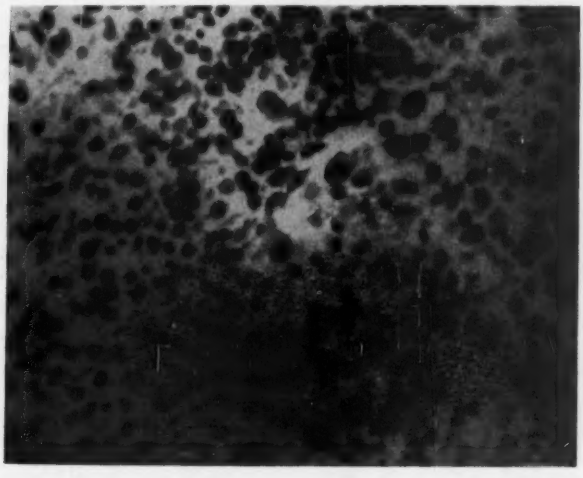

Fig. 1.—Lymphocytes and plasma cells with areas of necrosis and hemorrhage. A few foreign-body giant cells are seen.

under a generic term of histiocytosis X by Jaffe and Lichtenstein[6] and later by Mallory.[8] Primarily, they are nonlipid reticuloses. The appearance of lipid in Letterer-Siwe and Hand-Schüller-Christian disease is reported only in late stages.[2]

The eponyms by which these conditions are described seem good for clinical purposes but suggest little difference in the basic pathologic process.

These skeletal reticuloses are the nonlipid storage disorders of the reticuloendothelial system, the exact etiology of which has been a matter of discussion all these years. Thannhauser[11] believes that these xanthomatous formations are due to enzymatic disturbances, particularly in the young and proliferating reticular cells, and that they have a relationship with Gaucher and Niemann-Pick disease. Perhaps it may be related to Tay-Sachs disease also.

Hand-Schüller-Christian disease is a syndrome characterized by (1) osteolytic lesions, (2) exophthalmos, and (3) diabetes insipidus.

The exophthalmos is the result of a deposition of fat in the retrobulbar space, and diabetes insipidus is the sequela of pituitary involvement. The syndrome is generally found in adults and runs a very chronic course, but when it occurs in young persons it proves rapidly fatal. Visceral involvement is not uncommon, and pulmonary fibrosis is occasionally observed.

Letterer-Siwe disease, in contrast, is rare over the age of 2 years. It runs a rapidly fatal course, marked by skin eruptions, hepatosplenomegaly and lymphadenopathy.

Eosinophilic granuloma differs from the other two members of this family in that it is generally a solitary osteolytic lesion without any visceral involvement or any other evidence of invasion of the reticuloendothelial system. A high incidence is seen among children and in adults below 21 years. It also has a tendency to affect the skull bones frequently, which explains its higher incidence in otolaryngologic practice.

The Clinical Picture

Children and young adults are usually affected. Granulation tissue in the external auditory canal with serosanguinous discharge, or a soft swelling in the vicinity of the pinna, is the commonest way in which a case presents itself. The child is generally too well for such a lesion. The lesion may by its extent cause deafness and even affect the speech in very young children. Involvement of the middle ear, facial canal, and labyrinth is unusual, according to Dingley,[4] but facial paralysis has been recorded by Dawes.[3] According to Dingley, the lesion starts primarily in the squamous part and spreads to the mastoid bone, whereas Dawes states that the lesion may start either in the petrous or in the squamous part.

Other skeletal lesions may also occur in the ribs, pelvis, and calvarium, isolated or in association with the temporal bone.[5] Solitary lesions in long bones like the humerus and fibula, complicated by pathological fractures, have also been described.

There may be a slight elevation of the temperature, leukocytosis, and eosinophilia. Blood cholesterol, serum phosphates, calcium, and proteins are unaffected.[3]

Radiological Diagnosis.—The characteristic finding in this disease is a punched-out area in the squamous part of the temporal bone (Fig. 2). When the petrosa is affected, necrosis and marked disorganization of the bone is noticed. At times, a part of the petrosa may be seen dislocated from the parent bone.

Diagnosis.—The diagnosis can be confirmed only by a biopsy. All the characteristic histologic features may not be present in a particular case, and variants are seen which blend with other conditions of this category. A solitary osteolytic lesion with no systemic disorder or visceral involvement is indicative of eosinophilic granuloma. However, one must also consider the possibilities of tuberculosis, secondary metastasis, multiple myeloma, and chronic osteomyelitis in the differential diagnosis.

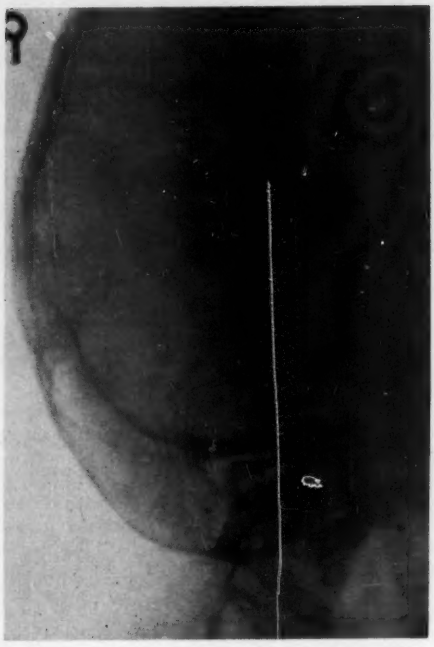

Fig. 2.—X-ray of the mastoid. Large punched-out areas are seen in the squamous and mastoid region.

The sites of the osteolytic lesions are similar to those of multiple myeloma and secondary carcinoma, and as the x-ray appearances may also be similar, mistakes in diagnosis readily occur.

In Dawes' series,[3] acid-fast bacilli were detected in the histological examination of one case, causing a confusion for some time. Examination for acid-fast bacilli and a pulmonary lesion are essential. Rarely, eosinophilic granuloma itself may be responsible for fibrotic lesions in the lungs,[1,2] and this may add to the confusion. A Mantoux test may prove useful in many cases.

Course and Prognosis.—Solitary lesions of eosinophilic granuloma have a tendency to heal spontaneously, and a good prognosis may be expected. Deafness, however, remains permanent. Revival of the labyrinthine function cannot be hoped for if once it has been involved. In acute febrile cases, where diffuse involvement of soft tissues and skeletal system has occurred, the prognosis is poor.

Treatment.—Deep x-ray therapy has been the treatment of choice and, when employed, a dose of 400-1,000 r has been adequate in most cases. Dawes, however, states that deep x-rays should be reserved only for painful and chronic lesions, since most lesions will heal spontaneously without any treatment.

Cortisone has been suggested[4] in the treatment of this disorder, but reports of clinical trials with this agent are not yet available.

Report of a Case

A 7-year-old boy attended the hospital for dull and intermittent pain behind the right ear for one month and attacks of giddiness not associated with nausea and vomiting. There was no history of any ear discharge. The child had had his tonsils and adenoids removed one year earlier.

On examination, there was a tender swelling in the right mastoid region. The right external auditory canal showed sagging of the postero-superior wall and slight congestion and bulging of the tympanic membrane, with no perforation or discharge. The left ear was normal.

Rinne's test was negative for the right ear, and Weber's test showed lateralization to the right side. However, no deafness was complained of by the child.

On a clinical diagnosis of mastoiditis, x-rays were taken. They showed large punched-out areas in the squamous and the mastoid region (Fig. 2). X-rays of the other skull bones revealed no abnormality. Hemogram was normal. Blood cholesterol, protein, phosphates, and calcium levels were within normal limits. Urinalysis showed no abnormal findings.

He was operated on under general endotracheal anesthesia by a postauricular incision. When the mastoid was explored, masses of pale, fleshy granulation tissues were seen filling up the bony defects. These were scooped out completely, and a conservative mastoid operation was performed, since there was no extension to the middle ear. There was little bleeding. The wound healed by first intention.

The biopsy specimen of granulation tissue revealed that the lesion was an eosinophilic granuloma.

Deep x-rays were started on the 10th day, and a total dose of 1,000 r was given to the mastoid region.

The child returned for a follow-up after six months and is in excellent condition.

Comment

The case differs from other cases so far reported in literature in that there was absence of ear discharge, and there was no other clinical finding suggestive of a granuloma. The tentative diagnosis of eosinophilic granuloma was entertained because of the punched-out areas in the x-rays of the temporal bone.

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Reconstruction of the Antihelix and Scapha in Selected Cases of Protruding Ears

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There is no one technique that is suitable for the correction of all types and degrees of protrusion of the external ear.* One must select a technique for the individual case, which in view of the bewildering array of possible techniques recommended in the literature is a difficult task.

Reconstruction of the antihelix and scapha is indicated where an underdevelopment or absence of both is the main cause of the ear deformity.

Where the concha is overdeveloped or the mastoid bulla pushes the ear outward, Sercer's technique of resecting the anterior spine of the helix, thinning the back of the concha, and excision of an ellipse or myrtle-leaf piece of retroauricular skin is well worth consideration. One should, in such a case of ear protrusion also consider Robin and Berlendis' approach to the cartilage incision in the incisura and also consider the possible reduction of the outer plate of the bone of the mastoid bulla.

In cases where the concha and mastoid bulla are markedly hypertrophied and where, in addition, there is underdevelopment of the antihelix and scapha, a combined procedure, such as that recommended by Vaheri, would seem to be the proper approach.

This group of cases, the most extreme of the protrusion of the ears, offers a surgical and artistic challenge that demands the utmost in skill and ingenuity. We are unable to standardize a procedure for these cases and are still evaluating various techniques.

This paper is concerned with *moderate* protrusion of the ear due particularly to the absence of a well-developed antihelix and scapha. Our technique is also applicable where there is *moderate* overdevelopment of the conchal cartilage.

The modern concept of the reconstruction of the antihelix stems from the classic contribution of Luckett, in 1910. His departure from the existing techniques is primarily that of locating the skin and cartilage excisions laterally on the auricle, rather than in the retroauricular sulcus, where the previous techniques had concerned themselves (Morestin, Monks, etc.).

Since 1910, two other articles have played a major role in the surgery of the antihelix. Davis and Kitlowski (1937) described the use of needles passed through and through the auricle to mark the antihelix. They recommended dipping the needles in a dye and then marked the cartilage with a series of puncture points to assist in the incision and modeling of the auricular cartilage.

New and Erich (1940) described a technique of external stay sutures that permitted an easy and flexible method of modeling the antihelix fold and maintaining the infolding of the cartilage in position during the period of healing. In 1955, Erich modified this technique somewhat, but we shall comment on his modification later.

In addition, to date, there have been a large number of articles concerned with the reconstruction of the antihelix, with many modifications and variations indicating a continuing search for artistic perfection.

Submitted for publication March 26, 1959.

* We agree with McEvitt (1947), who states, "A frequent mistake of the laity and even the profession is to refer to these cases as lop ears. Lop means to hang limply and the protruding ear certainly does not hang and is anything but limp."

The technique described in this paper is not a radical departure from all other techniques for the reconstruction of the antihelix. Rather, it attempts to emphasize a few aspects in which it differs from some while borrowing from others. The only originality is the clinical observation of the tendency of the auricular cartilage in the region of the helix to curl backward when it is incised near the free edge of the ear and when it is separated from its anterior lining perichondrium. This may be due to an embryologic shortening of the anterior skin of the ear in this region as compared to the posterior skin. This curling effect permits a soft roll to the antihelix when it can be used advantageously. In cases of moderate protrusion of the ear this soft roll will remain. In marked cases of protrusion, if more cartilage is excised in this region, a point is reached where the curling ceases. A sharper edge is then noted, but this may be necessary in order to bring the ear closer to the head. With further study we may be able to save this soft roll of the cartilage and bring the ear close enough by the other techniques mentioned before, i.e., Sercer's technique, Robin and Berlendis', etc. This may have to be done in two procedures because of the necessity of two incisions and the problem of blood supply of the intervening strip of skin.

In analyzing the operative techniques recommended for the correction of the antihelix, one can group the issues and discuss their merits as follows:

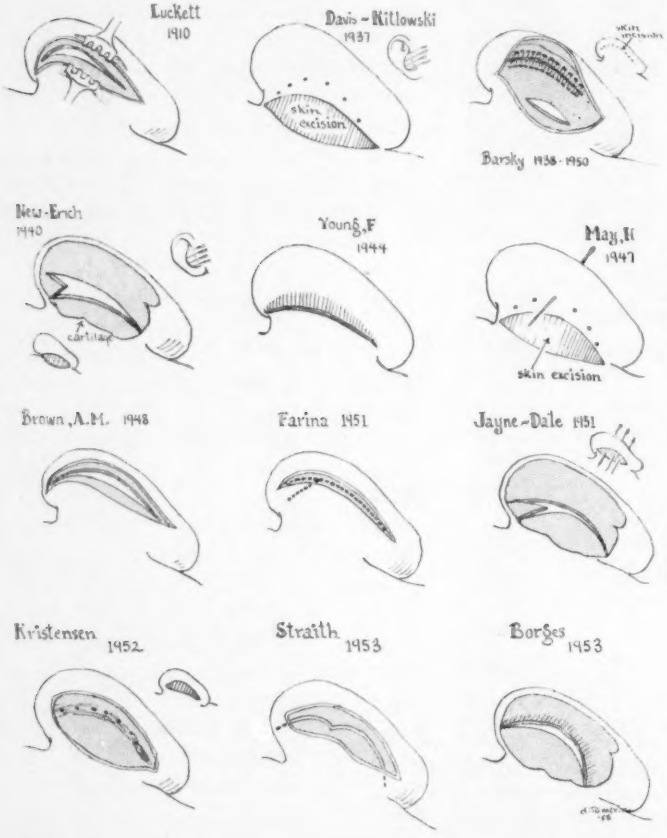

Fig. 1.—Diagrammatic review and comparison of various techniques for the reconstruction of the antihelix. The location and extent of skin and cartilage incision and excision is drawn as closely as possible to the description given by the various authors in their articles. The cartilage is shown stippled. One notes that there is marked variation in the place where the marking needles are inserted.

1. Where should the skin incision be made?
2. How much, if any, skin should be excised?
3. How does one determine the site where the cartilage should be incised and possibly excised?
4. How should the cartilage be modeled?
5. How should the modeled cartilage be fixed in its desired position? By internal or external sutures and how applied?

Where Should the Skin Incision Be Made?

Luckett (Fig. 1) made his skin incision laterally on the ear. Davis and Kitlowski (1937) and New and Erich (1940) made their skin incisions in the cephaloauricular angle. Young (1944) decried this cephaloauricular skin excision and recommended that the incision be made in the scaphoconchal angle and that the skin excess be removed from the lateral margins of the wound. Other techniques are shown in Figures 1 and 2.

For the selected cases of moderate protrusion of the ear where the problem is that of the underdeveloped antihelix and scapha and only partially that of the concha, we prefer a lateral incision made parallel with the rim of the ear about 1 cm. from its free border.

With the free edge of the ear as a guide one can make a mark with a dye along the ear to mark the location of the incision. One may also use a draughtsman's measuring pen to facilitate symmetrical marking of both ears. This incision should extend to the top of the ear and, if necessary, in front of it, as recommended by Hatch, if the ear needs to be drawn closer to the

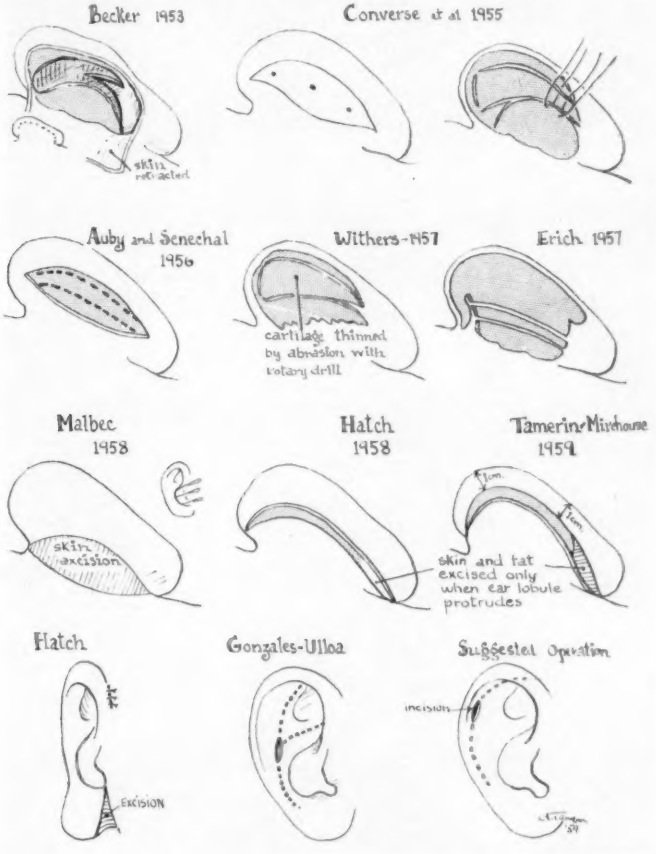

Fig. 2.—Diagrammatic review and comparison of various techniques for the reconstruction of the antihelix (continued from Fig. 1).

head at the upper portion. This need not invariably be done but is part of the technical armamentarium. This anterior prolongation of the scar may be obvious and sometimes, unfortunately, keloidal, but the risk must be taken, at times, if the postoperative "telephone deformity" of the ear as described by Gillies and Millard is to be avoided.

The incision may be continued inferiorly on to the fleshy ear lobe, where it can end or proceed around to the anterior region of the juncture of the ear lobe to the cheek. Here it permits bringing the ear lobe closer to the face (Hatch) and corrects the other end of the "telephone deformity" of the ear operated on.

How Much, If Any, Skin Should Be Excised?

A great deal of the skin may be taken without serious effect on the shape of the ear from the cephaloauricular region. Small amounts of skin removed laterally near the helix, however, markedly affect the shape of the ear and distort the free border of the helix, causing buckling, infolding and altogether remarkable variations in shape and contour.

It is wise to excise less skin rather than more, since any excess of skin is liable to shrink and at worst forms a lumpy rather than a smooth suture line behind the ear, where it is not particularly noticeable in any case.

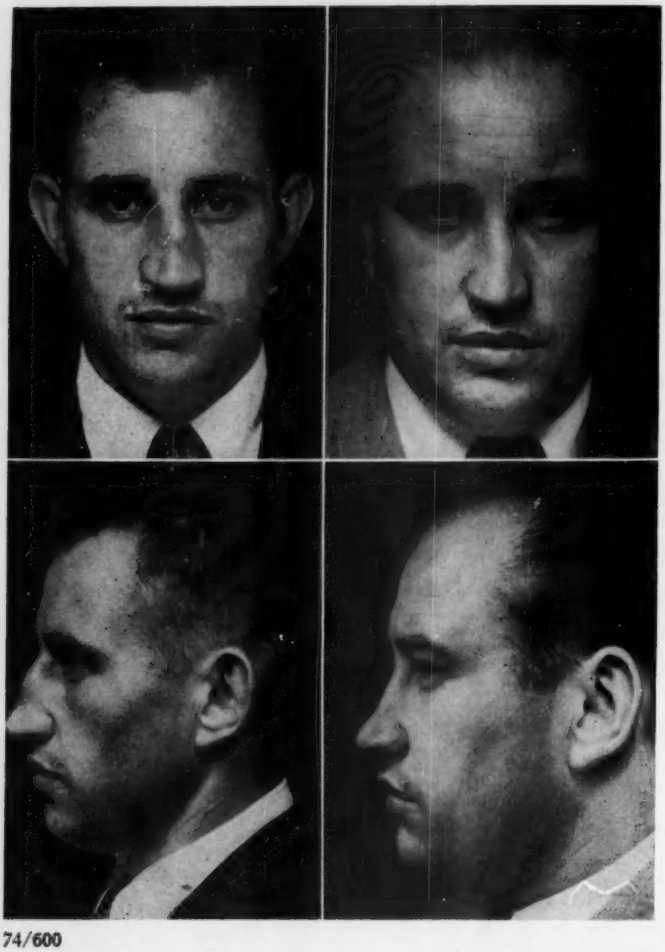

Fig. 3.—Result of otoplasty by our technique. This patient presented himself primarily for the reconstruction of a recent traumatic nasal deformity. One notes that the protrusion of the ear is primarily in the upper part. The antihelix is not flat but the helix needs to be drawn closer to the head. The concha is not enlarged and the ear lobe is close to the head. Such a case is easily corrected by our technique and, as can be seen in the lateral view, a rounded antihelix results.

How Does One Determine the Site Where the Cartilage Should Be Incised and Possibly Excised?

Luckett made the incision of the cartilage through a posterior approach fairly laterally on the ear but aside from locating it in the general region of the antihelix gave no specific instructions for placing the incision or for duplicating it on both ears. Davis and Kitlowski (1937) introduced the skin and cartilage marking with dye-tipped needles passed through and through the full thickness of the ear. This method has been widely copied. There is, however, wide divergence in opinions of where these needles should be inserted and what constitutes an antihelix.

Davis and Kitlowski, Young, May, New and Erich, Malbec, etc., place these needles at the medial margin of the antihelix and make their incision correspondingly more mesial than the incision recommended by Luckett. Some continue this marking in the direction of the inferior crus of the antihelix, while others (Young, Borges, etc.) mark the superior crus as well or instead of the inferior crus. Others, like Brown and Farina, make their incision quite far laterally.

Becker, Withers, Feuerstein, and Converse mark the cartilage and incise it in both lateral and mesial margins of the antihelix and model the cartilage from that point, by tubulization, in the instance of Converse, Withers, Senechal, Barsky, etc., and by folding over in the technique of Becker and others.

Gonzales-Ulloa incises the cartilage in what would correspond to the anterior crest of the antihelix, making a small external (lateral) rather than the usual posterior incision.

The incision that we employ exactly parallels the incision in the skin. It need not be marked by through-and-through needles, since it parallels the rim of the ear about 1 cm. from the free border. It terminates in the cleft between the tail of the helix and the concha. The tail of the helix is almost always discarded, and when it is, the helix falls backward against the head.

The incision is carried superiorly to the very upper margin of the cartilage much more anteriorly than the skin incision. Retraction of the skin permits the cartilage to be cut up to the anterior spine of the helix.

How Should the Cartilage Be Modeled?

The helix is now completely loose, and with the head in the lateral position, the helix will assume a new position by the force of gravity. One can easily see where the ear continues to protrude and the cartilage can be whittled away. The helix will correspondingly fall closer to the head. The posterior perichondreum is separated away, though it can be excised in an area corresponding to the cartilage excision. The anterior skin and perichondrium are elevated in one piece from the anterior surface of the cartilage for a distance of 1 cm. from the free edge. When this is done, the cartilage will be seen to curl backward and assume the shape of a more normal antihelix. This curling backward is particularly noticeable if the ear cartilage is thin, less so when it is thick, but is accentuated if the cartilage is shaved or thinned by abrasion with a rotary wire brush. The curl may also be accentuated by feathering the edge of the cartilage with a knife, a technique described by Borges but used in a somewhat different fashion.

Before the edge is feathered, or thinned by shaving or abrasion, the free edge of the cartilage (not that remaining in the helix) is whittled down. In the region of the antihelix, the curling of the cartilage continues for a distance of almost 1 cm. in many cases. As the excision approaches the conchal rim the curling of the cartilage ceases, and here the cartilage is excised as desired to permit the helix to fall backward into a desired position. This must be watched as the cartilage is whittled and can be recorded for comparison with the opposite side by measuring the distance of the

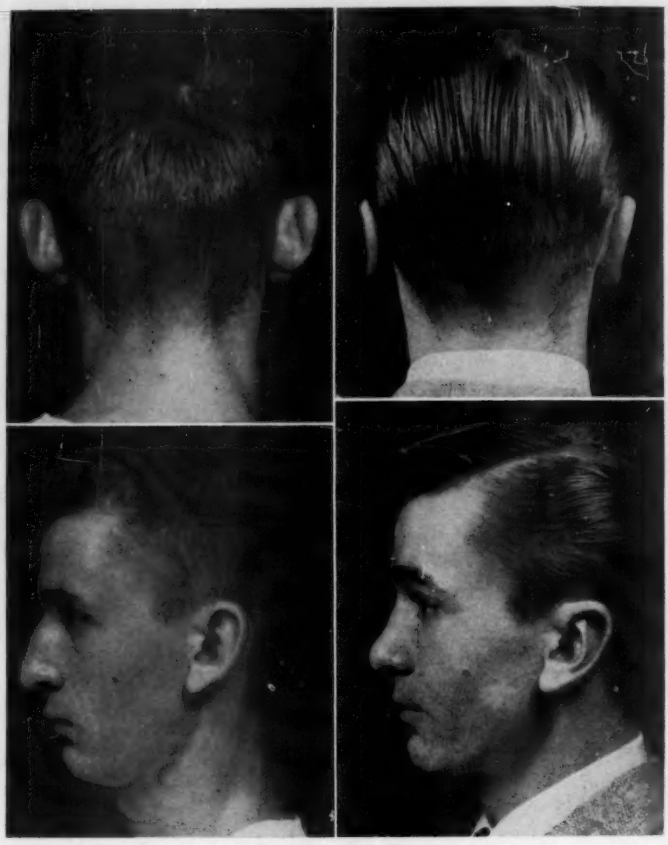

Fig. 4.—Result of otoplasty and combined rhinoplasty and genioplasty. The ears were brought close to the head, but the lateral view shows a sharper than desirable antihelix and a scapha that is wider than the ideal. This patient was entirely satisfied with the result and offered no complaint of any kind about the configuration of the antihelix. It has been our experience that patients do not complain of the antihelix sharpness if the ears are symmetrically close to the head and do not protrude unesthetically either at the top or bottom. It seems to us that the search for the rounded antihelix has been sparked by the surgeon's search for artistic perfection rather than by the patient's insistence.

free margin of the ear from the head. Unless one can judge by eye alone, it might be a good idea to record this distance at the top of the ear and at predetermined spots, i.e., middle of ear, level of antitragus, etc. We have not found that the estimation of the angles between the head and the ear or between the concha and the scapha have helped in the technique of this procedure. Rather trust one's artistic judgment or note the distance from the head to the free margin of the ear and record the pre- and postoperative difference.

As the helix falls backward and the protrusion is corrected, a scapha is formed that is of good depth. The width of the scapha will depend upon how much cartilage is excised. The bottom of the scapha will contain no cartilage, but its side walls will contain cartilage of the helix on the lateral side and cartilage of the antihelix mesially.

We should like to point out that in addition to a sharp antihelix, an abnormally wide scapha is deforming. As a matter of fact, a sharp antihelix is not noticeable if the lateral margin of the helix is well developed and the scapha is narrow and deep (Fig. 5).

Sercer complains that there have been insufficient lateral views of the results of otoplasties for protruding ears. He shows a poor result (not his own case) where the cartilage must have been cut mesially and the antihelical edge is sharp and unnatural and the scapha is abnormally wide.

The shape of a satisfactory scapha is not easy to describe. It will depend on the shape of the helix, which is extremely vari-

Fig. 5.—Result of otoplasty by our technique. One notes that a fairly sharp antihelix is here concealed by a narrow, deep scapha, as contrasted to the wider, shallower scapha in Figure 4. This patient and his family were entirely satisfied with the result. During the process of whittling down the cartilage in this case, the antihelix was rounder but the ear still protruded more than desirable. If the antihelix were left in this stage of correction and the balance of the protrusion corrected by Sercer's technique, a more esthetic lateral view would probably have resulted (Vaheri).

able, and the general shape of the ear. In general a narrow scapha is more esthetic than a wide one.

How Should the Modeled Cartilage Be Fixed in Its Desired Position?

If the ear has assumed a satisfactory position with the head held on the side, then it is obvious that any sutures that are used to maintain the shape of the cartilage will not be inserted under tension and cannot cut through.

Because the effect of gravity is essential to the performance of this technique, we perform this operation with the patient lying down with the head on the side, the ear uppermost, rather than with the patient sitting up (Malbec, Brendler, etc.). We have found no necessity for inserting internal sutures to maintain the cartilage in its new position.

The New-Erich external mattress sutures tied loosely (and without gauze rolls) are essential, however, to the technique. When they are inserted in two or three places, namely, between the scapha and concha below and between the scapha and fossa triangularis above, the ear assumes a quite

satisfactory shape. Where there has been no necessity to whittle a great deal of cartilage, a soft roll of the antihelix will result, forming a convex mesial wall of the scapha.

This technique has been most satisfactory in moderate protrusion of the upper ear. It has been satisfactory in more marked cases, though here one gets closer to a sharp antihelix border and one needs to cut more cartilage from the rim of the concha and even a part of the antitragus. In many such cases there may be a prominent lobule. This must be corrected by skin and subcutaneous tissue excision and even by a wedge of skin excision anteriorly at the junction of the lobule and the cheek, as recommended by Hatch.

The New-Erich mattress sutures have given us no skin ulcerations, even though left in for 10 days. We have therefore not seen the necessity of changing this method, as recently suggested by Erich (1955).

We insert these mattress sutures very loosely before the skin excision and skin closure. These mattress sutures maintain the ear folds in position against any influence of the skin closure. The amount of skin to be excised can now be judged easily and trimmed accordingly. The external wound can be closed as one wishes. Excision and closure of the wound before insertion of the mattress sutures is often very difficult. Fairly small amounts of skin and variations in the method of closure may produce some very remarkable variations in the shape of the helix. Such variations, often quite disturbing, can be obviated by establishing the desired shape and depth of the fold of the ear and the shape and depth of the scapha first by the mattress sutures and then by the skin excision and closure.

The resultant scar is in the bend of the antihelix and is generally very unnoticeable. We have seen three keloids of the posterior scar following otoplasty for protruding ears. Our percentage of keloidal thickening is higher than that of Patterson (1957).

Other details of techniques are as follows: Local anesthesia of 1% procaine hydrochloride (Novocain) with epinephrine is used by infiltration. Alphaprodine (Nisentil) given intravenously about two minutes beforehand dulls the pain of the local infiltration.

The entire head is soaked and painted with aqueous thimerosal (Merthiolate) 1:1,000. The hair is not shaved but is rendered sterile by the following method: The neck and face are painted with aqueous thimerosal with the patient sitting up. The hair is wet with aqueous thimerosal warmed in a basin of hot water. The back of the table is draped with a sterile sheet. The patient is directed to lie down with the head on to a sterile tray on which a towel soaked in aqueous thimerosal has been placed. The towel is wrapped around the head and is then soaked with a large bulb syringe filled with the thimerosal. The sterile metal tray prevents the soaking through and hence the unsterilization of the operative draping of the head. The thimerosal may be basted repeatedly on the hair through the towel for some 10 minutes or more. The alphaprodine is then injected with the patient recumbent, and the rest of the body is covered with sterile drapes.

We have been able to use local anesthesia in children as young as 8 years but do not hesitate to use endotracheal anesthesia whenever necessary.

With the hair sterilized for the purposes of this operation, there is no problem of turning the head from side to side to compare the shapes of the ears following surgery. Bulky drapes applied to the head, where the hair is not rendered sterile, are difficult to manipulate. They may come loose and may permit unsterile hair to slip into the operative field and prevent easy comparison of the ears for asymmetries. By our method, if at the end of the operation one is not sure of the result, one can have the patient sit up without unsterilizing the field and compare the two ears from both in front and in back. If necessary, one or both sides may be further modeled. Prominence of the lobule and/or

the upper part of the ear is more favorable with the patient sitting up, and at this [redacted] such protrusions can be estimated and the necessary adjustments made by excision of more skin, etc.

When one is satisfied that further surgery is either not needed or should be postponed, a moderate pressure dressing is applied. No drains are used. Hemostasis should be meticulously achieved during the operation. Under local anesthesia, many operations are practically bloodless. If the dressing is applied too tightly, pressure necrosis of the ear or forehead skin may result. One should leave specific instructions that if the patient complains of tightness and excessive pain the dressing should be loosened *at once*. The dressing is left on for a few days. The mattress sutures are removed in 10 days. The ear is kept against the head by a stocking cap or headband. During the day, when such a dressing is awkward, the upper part of the ear can be kept close to the head by gluing it to the skin of the mastoid region with rubber cement. The use of the rubber cement may be useful for a matter of weeks following the operation. Skin dermatitis, if it ensues, is easily managed. The rubber cement is discontinued and the ear is left exposed to the air as much as possible. It can often be resumed. We have seen cases that were saved from secondary correction by the long-time use and training of the rubber-cement adhesion.

Summary

Various techniques from the literature for the modeling of the antihelix in the correction of protruding ears are presented, with particular reference to their divergence from the Luckett operation from which they stem.

Our operation, closely approximating that of the Luckett operation, consists in the incision of the skin and auricular cartilage parallel with the free border of the ear about 1 cm. from its edge. The cartilage is completely freed from the spine to the tail of the helix, which is often excised. [redacted] anterior perichondrium is [redacted] the medial border of the incised cartilage, the cartilage is found to curl backward. This may be due to a congenital shortness of the anterior skin of the external ear in the region of the antihelix. This curling produces a rounded antihelix which in moderate protrusion remains after the correction. If markedly protruding ears, particularly those where the concha is enlarged, are corrected by this method, a sharper antihelix and a wider scapha result. This result is less artistic but not, in our experience, objectionable to the patient. In such cases, our method combined with Sercer's technique may be preferable.

44 E. 67th St. (21).

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Discussion of the Neurochronaxic Theory (Husson)

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Raoul Husson started a veritable revolution in 1950 [1] as far as the physiology of the vibration of the vocal cords is concerned. He did not merely attempt to change some of the previously accepted opinions but proposed a basic change of nearly all concepts which up to that time had been considered valid.

The discussion of this completely new approach to vocal physiology has become a widespread international matter, but the American literature has not taken up the subject in its full breadth, and only some stray assents and objections have been published.[2,3] Although I was opposed to this trend of thinking from its beginning,* I shall try to characterize the approach of the Husson school with the greatest possible objectivity and then bring forward the arguments which oblige us to reject the contention of Husson. His basic hypothesis consists in supposing that the vocal cords contract actively and open the glottis in the frequency of the vibration, the closure of the glottis being effected by the elasticity of the vocal cords; the subglottic pressure and the flow of the air through the glottis have no dynamic influence on the vibrations of the vocal cords.

Husson calls his theory "neurochronaxic," and we do not want to dispute his right to do so. His denomination of the classical theory as "myoelastic" is completely insufficient and a misnomer because Husson's hypothesis is also "myoelastic," as it attributes one phase (opening) of the vibration to muscular contraction and the other (closure) to forces of elasticity. The expression "myoelastic and aerodynamic" is more correct, and, for the purposes of this discussion, the simple expression "aerodynamic theory" is fully satisfactory.

Submitted for publication Feb. 7, 1959.

* I want to emphasize this because the circumstance that Husson had warmly dedicated his basic publication to me—as well as to his professors and collaborators in Paris—might lead to some misunderstanding.

I †

Husson presented in 1957, as the main foundations of his views, the following arguments [4]:

Goerttler [5] disproved the generally accepted view that the vocal cords consisted of longitudinal muscle fibers. He showed that the vocal cords consisted of two strands of muscle fibers, which originated at the thyroid and arytenoid cartilages, respectively, and approached the free edge of the cord in an angle, terminating in the ligamentum vocale (Fig. 1). The contraction of these muscles pulls the vocal cords laterally, thereby opening the glottis; this is the opening phase of vibration. The closure of the glottis is effected by the elasticity of the tensed vocal cords them-

† This section is an exposition of the views of Husson. The detailed discussion of these arguments will follow in Section IV.

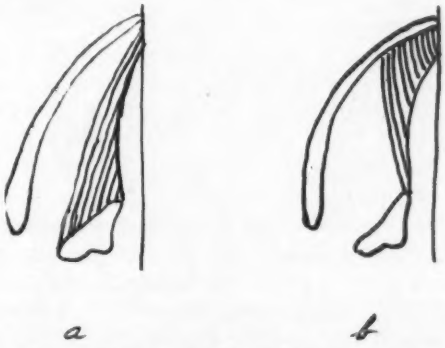

Fig. 1.—The two strands of the vocal muscle, the musculus aryvocalis and the musculus thyreovocalis, following Goerttler.[5]

selves after the lateral pull subsides. Consequently, we should not speak of "vibration" of the vocal cords, since this would suppose a "movement maintained by an elastic automatism." As the decisive phase of the movement is an active one, i.e., due to a muscular contraction, the larynx does not "vibrate" during phonation, but it presents a kind of shutter mechanism ("obturateur").

Laget [6] and others stimulated the recurrent nerves of dogs with electric currents of high frequency. At up to 600 per second, they obtained vibrations of the vocal cords which were synchronous with the frequency of the electric stimulation. By previous tracheotomy, the influence of the streaming air was completely excluded. This showed that the vocal cords can respond with this high frequency and do not need any air for their vibrations; these latter are due to distinct stimulations of the recurrent nerves to which they answer "blow by blow" (*coup par coup*).

Moulonguet [7] and others applied electrodes to the recurrent nerves of three human subjects (whose neck was opened for a subsequent operation) and were able to find electric currents of the frequency of the sounds produced by the larynx. This furnished the evidence that the recurrent nerves of man carry electric-action currents in the frequency of the so-called vibrations of the vocal cords.

Portmann and Robin [8] made a similar investigation on the muscles of the larynx by methods of electromyography. They found signs of electric activity in the muscles which corresponded in their frequency to the sound produced simultaneously by the larynx.

Coraboeuf et al.[9] showed that fibers of the recurrent could, when appropriately stimulated, double their phase of response. This tied in with the contention of Husson (1950) that, in order to arrive at higher and highest frequencies of the human voice, the action of nerve and muscle fibers would proceed with a phasic difference. At first, only one group of them would respond, then, in the refractory interval of the first group, another group, finally a third and fourth group would react in a staggered manner. These changes to a biphasic, triphasic, etc. function would then constitute the change of registers.

Kirikae, cited by Husson,[4] found in his stroboscopic studies that the opening of the glottis (during phonation) started in a descending direction. If the opening phase would be due to the pressure of the subglottic air, the initial movement should be an upward one.

Piquet and his collaborators [10] demonstrated a high-frequency cinematographic reel which showed a patient (about to be laryngectomized) whose upper respiratory system was cut above and below the larynx, but the connection of his recurrent nerves was preserved. They tried to exclude any air coming through the sectioned trachea and asked the patient to phonate. Perfectly normal-appearing vibrations of the vocal cords could be seen. This convinced Husson that the air does not play any dynamic role in the so-called vibrations of the vocal cords, and he offered a mathematical formula to back up his thesis. The only role which could be attributed to the subglottic pressure is, consequently, merely a strong sensory influence on the subglottic walls of the larynx, heightening the excitability of the muscles in a proprioceptive way.

Garde, cited by Husson,[1] made stroboscopic observations on Husson's vocal cords and noticed that, during the singing of the same note, the vocal cords showed a smaller amplitude in forte (loud) than when singing piano (soft). Also, Timcke [11] has established the same rule, which contradicts the dynamic role of the air in the production of vocal sounds, because in this case the increase of subglottic pressure should result in a greater amplitude of the vibrations. The tomograms taken by Husson and Djian [12] show a broader contact of the swinging vocal cords (in the phase of closure) when the subglottic pressure in-

creases, contradicting any possibility of the dynamic role of the air stream.

This certainly seems to be an important array of proofs, especially for those who could be baffled by either anatomical, physiological, neurophysiological, stroboscopical, or acoustical arguments. However, the above arguments have been subjected to a careful scrutiny by the respective experts and the result is negative. I shall not forget in this discussion the above points, which Husson seems to consider the most important ones, and especially the basic question: Which is the role of the airflow in the production of laryngeal sounds?

II

This is the third attempt, in this century, to prove that the vibrations of the vocal cords are due to active contractions in the frequency of the sound produced by the larynx. Stern [13] demonstrated that in many cases of pathological phonation one of the vocal cords frequently stood still at stroboscopical examination. He took this as an indication that each vocal cord could be active independently; this implied that the vibration of the vocal cords was due to a process of active contraction. Weiss [14] was able to show that the apparently immobile vocal cord was swinging with a different frequency and, therefore, notwithstanding the correctness of the observation itself, the conclusions of Stern were inadmissible. After a lively and generalized discussion (summarized in Reference 15), even Stern had to admit that he fell victim to a stroboscopic misinterpretation.‡

Heymann [17] described, in 1933, the stroboscopic observation of an active wave-like movement of the vibrating vocal cords, which started at the ventricles of Morgagni and ran toward the glottis; it disappeared at the free edges, which the stream of air made vibrate passively. He concluded that there was an active, i.e., muscular impulse in the vocal cords corresponding to the frequency of their vibration. This observation received less attention than it might have deserved, but Husson adopted Heymann as the forerunner of his ideas,[18] overlooking the fact that even in this short formulation the two conceptions are irreconcilable. (Heymann spoke of an active movement toward the median line, and Husson, of one in the lateral direction.)

Husson started on a broader basis; in fact, he later on tried to encompass the whole of voice physiology. It might not be fair to confront him with his earlier pronouncements, as even his conception of the active contraction of the vocal cords has undergone considerable changes. Lately, he has found it necessary to rather correctly assess the scientific status of his views in saying, "What we offer is not a theory; it is only a working hypothesis which we change corresponding to the emerging experimental facts." [4] This makes for extreme elasticity in more than one sense and is very different from the bombastic and authoritarian statements which characterized the publications of the Husson school in former years. We have, therefore, presented his latest views (as they appear in the literature) without historical recriminations and intend to deal with the cornerstones of his scientific structure.

Let us contemplate for a moment the attraction of the idea of the active contraction of the vocal cords as the cause of their vibration, which has made for its rather widespread, if superficial, acceptance. This conception obviates the necessity of the

‡ At this point it may be useful to remark that a stroboscopic examination of the vibration of the vocal cords, notwithstanding its undeniable value for clinical purposes, is subject to considerable errors in the scientific interpretation of the observed phenomena; the stroboscopic picture is due to an optical trick, and the resulting impression is a composite picture of many vibrations under suppression of the peculiarities of individual vibrations. This situation is somewhat improved by the new trend of electronic stroboscopy, started by Kallen and Polin,[16] but undiminished scientific criticism is necessary for the interpretation of the stroboscopic *appearance* which should not lightly be equated with the factual happenings in the larynx.

knowledge of extremely intricate (and often mathematically obfuscated) acoustical and mechanical laws and, at the same time, covers any possible gap in our knowledge by the *deus ex machina* of a voluntary action of our central nervous system. If the person "does it," we feel less responsible to find out the laws which made it "happen." A similar shift of responsibilities makes many people dump into the lap of the psychiatrist a great number of problems which should rather have been subjected to painstaking medical scrutiny.

III. General Objections

1. Husson postulates a vocal organ which, in both its peripheral and its central structures, would have been developed exclusively for the purpose of phonation. No other purpose could be served by the extreme frequency of contractions (up to 500 and, when functioning polyphasically [see above], as high as 2,500 per second). This contradicts the generally accepted view that phonation only makes particularly refined use of peripheral structures which were developed by nature for other and biologically more basic purposes.

2. The brain center which Husson postulates should be able to emit stimuli in the whole frequency range of the human voice up to 2,400 per second and more. Husson quotes as a parallel the demonstration of Stevens and Davis,[19] who showed that the central hearing can perceive vibrations of even much higher frequency. There is, however, a basic difference between the sensory and the motor system. The maximum frequency of the voluntary stimuli as a rule does hardly surpass 50 per second (some authors arrive at the extreme of 100 per second). However, Husson's conception also presupposes an absolute coordination of the centers on both sides and consequently needs a superimposed leading center.§ Such an extremely complicated structure, which is supposed to have been developed by man only and therefore a very late acquirement in philogeny, should be extremely vulnerable and among the first to suffer from a lesion of the brain. No such occurrences are known in the pathology of the central nervous system.

3. All the phenomena of the vibration of the vocal cord can be produced on the larynx of the dead (humans as well as animals) and in all kinds of experimental instruments. The pseudovoice of the laryngectomized can be developed occasionally into a well-sounding and even musical one. Thooris Van Borre[22] experimented on some subjects and described a circus performer who was able to "sing" musical pieces with his anal muscles after imbibing gaseous drinks. All these vibrations are produced without "active contractions in the frequency of the sound."

It was demonstrated on dogs that, notwithstanding the section of both recurrent nerves, normally vibrating vocal cords could be seen if the necessary constriction of the glottis and an appropriate pressure of the subglottic air were applied.

4. Every laryngologist is familiar with the surprisingly good voice of patients who have a bilateral paramedian position of the vocal cords, presumably due to a paralysis of the recurrent nerves. Froeschels[3] arrived at clinching this important point by collecting several reliable cases of bilateral paramedian position from the literature in which there was no important disturbance of the (speaking) voice, notwithstanding having found on autopsy that both recurrent nerves were completely degenerated. This definitely proves that, given a certain tension of the vocal cords and sufficient narrowing of the glottis, the subglottic air pressure can and will produce vibrations of the vocal cords. Why then should nature go to the trouble of developing completely

§ Husson tried to prove that the centers on both sides can function independently from each other and are connected with the homolateral vocal cord[20]; this was refuted by Kagen and Luchsinger.[21] This is mentioned only to show the theoretical necessity of bilateral centers and of a coordinating one, if we follow Husson's ideas.

new central and peripheral structures of unheard of complexity just to make for some refinement in phonation?

Our public and private discussions of these matters during the last years keep me convinced, however, that polemic questions directed to either side will not decide anything, because even in case they are answered, the responses will bear the stamp of the original conviction. We want to deal, therefore, with the validity of the arguments which Husson himself considers important.

IV.‖ Specific Objections

Goerttler.—The new description of the muscles of the vocal cords as consisting of two strains, which originate on the thyroid and arytenoid cartilages, respectively, and meet in an obtuse angle at the ligamentum vocale, evoked a lively discussion among the anatomists. Wustrow,[24] van den Berg and Moll,[25] Mayet,[26] and others tried to demonstrate that Goerttler made the mistake by getting into the subglottic area (conus elasticus) and that no fibers of the vocal muscles are fixed on the vocal ligament. This discussion is yet going on (Behringer and others[27]), but we want to try here to evaluate Goerttler's findings, taken at their face value, and decide whether they could be used as a backing for Husson's conception. (It should be noted that Goerttler has declared himself neutral in this discussion, emphasizing that his findings could be used by both sides.)

Two elements of this description of the muscles of the vocal cords seem to exclude their function as postulated by the neurochronaxic school:

(*a*) The two strands of muscles approach the vocal ligament at such an obtuse angle (Fig. 2) that the lateral displacement resulting from their contraction would be insufficient to account for the amplitude of the vibration of the vocal cords.

(*b*) Goerttler[5] describes a torsion of the vocal cords in the phonatory position (Fig. 3). (A somewhat similar torsion is described by his main adversary, Wustrow.) Such a torsion would, on the one hand, reduce even more the disposable muscle fibers which would pull the vocal cords laterally; on the other hand, the stiffening of the edge of the vocal cords by the torsion would further resist any such pull.

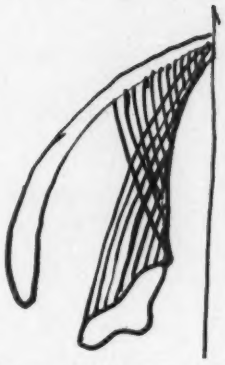

Fig. 2.—The basic structure of the musculus vocalis, following Goerttler[5] (superposition of Fig. I*a* and *b*).

Consequently, even the description of the muscles of the vocal cords by Goerttler would be unable to account for the frequency, and especially the amplitude, of vibration of the vocal cords. The theory of Husson lacks, therefore, the indispensable anatomical basis as far as the muscles of the vocal cords are concerned.

Laget.—In this experiment, high-frequency (100-600) stimulation of the recurrent nerve elicited responses in the vocal cord of a tracheotomized dog. Neither sound production nor flowing of air was involved. It came to us as a surprise that the muscles of the vocal cord could respond at such high frequencies. A number of authors in the past only got as high as

Fig. 3.—The muscular torsion of the free edge of the vocal cord in the phonatory position, following Goerttler.[5]

‖ Compare Section I.

50 per second, beyond which level the muscles fell into tetanic contraction. Dunker and Schlosshauer,[28] who tried to repeat Laget's experiment, came to the conclusion that there was no relationship between the frequency of the vocal cord vibrations of the dog produced by electric stimulation without airflow and that of the sound produced under the same stimulation; neither did they find any vibrations over 200 per second which could be registered or observed by stroboscopic means. Beyond that range only "muscle tone" could be found. They concluded that the resulting movements would have been too small to exert any influence on the production of a laryngeal sound anyway.

The registrations in the original publication of Laget also speak in favor of this latter conclusion. Also theoretically important is the description in Laget's experiment that the to-and-fro movement of the vocal cords (which we call vibration) always started by a narrowing of the glottis. This contradicts Husson's contention that stimulation of the recurrent nerves leads to an opening of the glottis.

The experiments of Dunker and Schlosshauer bring up another important point: The fact that some electric potentials can be imposed by stimulation of the nerves or muscles of the larynx, or that some spontaneous electric potentials can be recorded,[8] does not mean that they lead to distinct muscular movements or even decide the frequency of electric stimulation and the sound produced by the larynx. This means that whatever results Laget elicited, they are an experimental artifice without bearing on the basic question of natural phonation.

Moulonguet and Portmann.—The changes of electric potentials in the recurrent nerve corresponding to the frequency of the sound were first recorded by Lindemann,[29] on dogs. His explanation was (following Preisendörfer[30]) that they were proprioceptive stimuli originating in the swinging vocal cords and not motor stimuli originating in the brain. Husson asserts most emphatically[4] that such proprioceptive stimuli exist; only he does not say which way they take. This would, therefore, be one of the possible explanations of the observations of Moulonguet, as well as Portmann, who registered electrical potentials in the frequency of the larynx sound in the human recurrent nerves and vocal muscles, respectively.

Van den Berg,[31] the most efficient adversary of Husson, brought up the important point that such registrations might be due to a microphonic effect; purely mechanical oscillations (like those of the vocal cords) might transmit electric potentials in a nearby electrical system, without any primary electric stimulation being involved. Husson and his school objected to this interpretation, and we are unable to decide this question, which belongs to electroengineering. There is, however, an irrefutable dilemma involved in the following consideration: Husson is of the opinion that the neurochronaxic production of the larynx sound is an exclusive patrimony of the human species, and, consequently, he declines animal experimentation as proof or counterproof.[32] Lindemann,[29] however, showed that similar changes of electric potentials ¶ occur also in the dog; lately Paulsen[33] has found them in frogs. Therefore, Husson would have to extend his neurochronaxic theory into the animal kingdom or to admit that the frequency modulation registered on the recurrent nerves must be attributed to something rather unspecific, namely to proprioceptive stimuli or to the microphonic effect.

Coraboeuf.—It is of some theoretical interest that in these experiments fibers of the recurrent nerve could be forced into what seems to be a duplication of their

¶ It will be noticed that we try to avoid the historically correct but misleading expression "action potential." Such potentials are signs of electric changes but no proof that such changes are necessarily always due to spontaneous electric activity or leading to muscle *action*. It seems that some authors just habitually assume such a meaning and arrive at erroneous conclusions.

rhythm. This does not prove yet, however, that such is the case in the normal phonation. On the other hand, what Coraboeuf obtained was a strict alternation between stronger and weaker potentials, which is contrary to what we can observe in any kind of natural phonation.

Piquet and Others.—The demonstration that the vocal cords can vibrate even when all airflow through the glottis is excluded was certainly surprising and forced even Husson to change the basic tenets of his concepts (see above). But was the air really excluded? Dunker and Schlosshauer [28] tried to repeat the experiment by making sure that no air could pass through the larynx by pressing a glass plate against the lumen of the trachea: no vibrations could be seen. Van den Berg [34] called attention to the fact that the closure of the trachea by an inflated balloon (in the original experiment of Piquet et al.) might not have been really airtight, given the great pressure of the air in the trachea. Piquet and others [10] also very correctly mentioned in their original publication that there might have been some low moaning sound produced by the larynx which was completely drowned out by the noise of the various apparatus. An audible sound of the larynx presupposes airflow, also in the view of Husson et al. They deny the dynamic role of air, but its acoustic role they have not attacked, so far. This seems to prove van den Berg's objection.

Our main concern, however, is with the circumstance that nobody else was able to repeat the experiment of Piquet and others successfully. All known evidence contradicts the possibility of a vibration (or "vibration") of the vocal cords without airflow. Although it was long considered as a truism, a number of authors stepped forward to prove that if the tracheostoma was left open, or opened suddenly, no vibration of any kind could be observed at all—let alone such complete and complex vibrations as visible in Piquet's film. As long as this experiment cannot be repeated under reliably controlled circumstances, above all by excluding with definite assurance any airflow (like the glass plate applied by Dunker and Schlosshauer), we have to regard the experiment of Piquet and others as a fluke, due to a fortuitous passage of air, as indicated by van den Berg.

For those who are conversant with the more intricate problems of the vibrations of the vocal cords, the objection of Smith might be of great significance. The vibrations of the vocal cords on Piquet's film show the same phasic differences as could be demonstrated by earlier authors, as well as Muller [35] and Smith,[36] on experimental pipes whose vibrations were produced by the stream of air pressed through them. Starting with the initial position of broad closure of the vocal cords (seen in the "chest register" and estimated by Husson and Djian as capable of reaching the depth of 20 mm.), we can observe as follows:

(1) Complete closure of the glottis (Fig. 4*a*)
(2) Opening of the upper part of the closed glottis (Fig. 4*b*)
(3) Complete opening of the glottis (Fig. 4*c*)
(4) Medial swinging and closure of the upper part of the glottis (Fig. 4*d*)
(5) Closure of the glottis in its whole depth (Fig. 4*e*).

Such are the complex phasic vibrations visible on the film of Piquet and others; they cannot be explained any other way but that they were made to vibrate by the flowing air. Husson would have to add to his already very complicated basic conception yet another more intricate mechanism in the vocal organ, which would account for these phasic differences during a single vibration; and all this should have been developed by nature for the exclusive function of phonation?

Garde.—The observation of Garde on the larynx of Husson to the effect that the amplitude of vibrations of the vocal cords in singing loudly is smaller than when singing softly, contradicts all our respective experiences. The contrary has been observed by all authors who wrote about this subject.[37] What then is the explanation of Garde's finding? We can only venture an explanation because we have never been

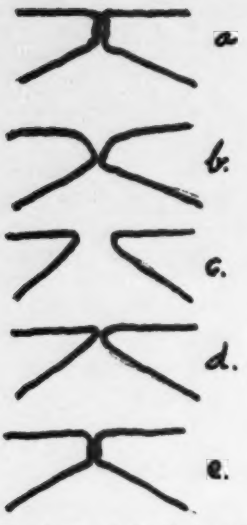

Fig. 4.—The phases of the closure of the glottis (chest voice). (*a*) Complete closure; (*b*) closure of the lower part of the glottis; (*c*) complete opening; (*d*) closure of the upper part of the glottis; (*e*) complete closure (disregarding the vertical components of the vibration).

able to verify his description. In conventional stroboscopy, as used in this experiment, the full amplitude of the vibration of the vocal cords can be seen when a practically constant pitch can be maintained by the singer. This is, in the case of most singers, easier when singing *piano* but difficult when singing *forte*. If the pitch of the voice deviates from that of the stroboscope (instead of maintaining the optimal 1-3 per second difference), be it by a strong vibrato or by plain false intonation, the amplitude "appears" reduced because of the stroboscopic procedure and not because it is actually smaller. If the difference becomes too considerable, the vocal cord might even appear immobile. (This was the explanation that was generally accepted as the explanation of Stern's observation of the immobility of one vocal cord during phonation.) This observation participates in the series of those experimental "facts" which were, for the first and last time, observed by the Husson circle but not found by anybody before or after; thus we can only surmise that Husson had stricken another false note—this time as an experimental subject.

Kirikae.—This experiment was not reproduced by anybody else; perhaps also because it requires the extreme and delicate skill of the Japanese experimentator. His experiment consists of applying a radiopaque material at the edge of the vocal cord, sufficiently small so as not to disturb normal phonation but big enough to be followed by stroboscopic observation. This certainly is an extraordinary feat of skill. Regarding his conclusions, however, serious doubts arise from the following circumstance. Due to the stroboscopic procedure, which requires a small difference (1-3 per second for sufficient observation) between the frequencies of the phonation and the stroboscope, respectively, it is often very hard to decide the sequence of events, namely, whether a certain movement pertains to the opening or the closing phase of the vibration. If the stroboscopic frequency is 1-2 per second lower than the frequency of phonation (this is the more or less ideal relationship), the opening and closing phase appear in the right sequence. If the stroboscopic frequency is only 1-2 per second higher than that of the vibration, the "apparent" opening phase is, in fact, the condensation of the closing phase and inversely; in simple words, the picture turns backward. It is due to the same optical illusion that in the movies (which have many similarities with stroboscopy) we can occasionally see the spokes of the wheels of running carts turn backward. (This means that their revolution per second is lower than the frequency of the exposition of the movie camera, which is usually about 16 per second. When they approach this frequency, they appear to slow down, and they seem to stop when they hit the exposition frequency of the camera).

This means that in stroboscopic observation the mere difference of 2 vibrations per second (inaudible for the observer and only detectable by a delicate experimental

set-up) can make the difference between a forward or backward running picture. We were not able to study Kirikae's publication in the original in order to be able to say whether or not he was cautious enough to avoid this pitfall. His observation has no decisive value in this discussion anyway, but it serves to illustrate the important point that all stroboscopic observations—as great as their clinical value might be in general—have to be subjected to the most painstaking criticism when used as a means of scientific validation of a given argument. What we see in stroboscopy are not "facts" but a certain summarization of repeated events by means of an optical trick. This, however, should not trick the observer into believing that what he has seen is necessarily the full reality.

Summarizing our objections against the most important pillars of the theoretic edifice of Husson, we come to the conclusion that the anatomic (Goerttler) basis is unacceptable, the physiological observations unique (cannot be duplicated by authors outside the Husson circle) and/or of doubtful value. Under scientific scrutiny, not a single one of the "proofs" proposed by Husson has stood up.

V

Before finishing this part of the discussion, we have to consider the extreme conclusion which Husson derived from the experiment of Piquet (who does not belong to the Husson school). Husson now supposes that the air has no dynamic role at all in phonation. (In earlier times, he allowed that the streaming air was responsible for the amplitude but not the frequency of the vibrations.) He offered mathematical formulae to prove that the forces of tension and elasticity are so much superior to the strength of the airflow through the glottis (10:1) that the latter becomes negligible and unimportant. The airflow is passively altered by the "vibrations" of the vocal cords and merely makes them audible; it is not necessary for their vibration and has no influence on frequency or amplitude of the same. This new contention is contradiction to so many well-known facts that we can only touch upon some of the most decisive ones.

The influence of the subglottic air pressure upon the frequency of the vibration of the vocal cords (pitch) was first (1837) demonstrated by the classical experiments of J. Müller[38] on the excised larynx of cadavers. He found that increase of this pressure led to a higher frequency; if he desired, therefore, to maintain the same pitch, he had to lower the tension of the vocal cords proportionally. He called this relationship the "law of compensation of forces in the larynx." This basic theoretical rule was taught everywhere, but there appeared inexplicable contradictions in the practice of singing. Weiss[39] summarized these contradictions in 1937 and was able to show that in the so-called head voice # the phonation follows Müller's law of the compensation of forces; the pitch rises parallel with the subglottic pressure. In the so-called chest voice, however, the pitch is lowered by the increase of subglottic pressure. This leads, on the one hand, to the disagreeable sharpening (rising) of the strained high notes of sopranos, and on the other hand to a painful lowering of the high notes of male singers who try to hit them by an excess of glottal closure and by strongly increased air pressure. These influences of the air pressure can be even more clearly demonstrated if the singers try to "overblow" those high notes when subjected to the "deafening" * by Lombard and Baldenwech,[40] because they are unable to compensate for the deviation resulting from an excess of air pressure.

Also possibly in cases of pathological phonation which, by the fact that the glottis could not be properly closed, rather resemble the head voice.

* This deafening procedure consists in the overloading of both ears with an instrumental noise which makes it impossible for the subject to hear his own voice. Thus, he is unable to control it by listening to it himself and, therefore, all acoustical phenomena can be studied without the interference of the subconscious tendency of correcting any deviation.

We have criticized above the "unique" observation of Garde (on Husson's larynx) which seemed to show that the amplitude of the vibrations decreases with the increase of volume of the sound. The observations of every author on the subject (compare the excellent review by Luchsinger [37]) prove the contrary, namely, the increase of amplitude with the increase of volume. This is strictly correlated with or—simply said—due to the increase of subglottic pressure.

Besides the incontrovertible fact that both frequency and amplitude of the vibration of the vocal cords are lawfully influenced by the air pressure, let us quote the elegant demonstration of van den Berg [41] with helium gas (which serves among others to fill balloons). This inert gas is of a much lower density than the air and when the subject intended to sing a certain note, this latter was considerably higher if he had inhaled a helium gas mixture previously. Individuals singing with a helium gas mixture in their lungs were able to increase the upper limit of their normal pitch up to six notes.

These few examples might prove anew the absolutely basic fact that the air is decisively important for the vibration of the vocal cords and this cannot be disproven lightly by an experiment of difficult and doubtful interpretation (the film of Piquet) or by a mathematical formula with a number of hypothetical elements, offered by Husson.

VI

Some hypotheses, even if they are not reliably backed up by facts, have the heuristic value of opening new avenues for our theoretical thinking or practical procedures; these advantages might remain even if the basic hypothesis should turn out to be indefensible.† Therefore, it might be of interest to consider which theoretical or practical (therapeutical) advantages might be derived from the neurochronaxic hypothesis.

As far as we can see, the neurochronaxic approach offers only one "advantage." It substitutes the complicated acousticomechanical explanations of phonation, which are so difficult to grasp for the student of these problems, by an oversimplified scheme of electrophysiology. Otherwise, however, we see only new difficulties in the way.

As far as anatomy is concerned, the Husson school has yet to find the necessary muscular organization in the vocal cords which could account for the extreme rapidity of the "active vibrations." The idea started out on the shaky basis of the discredited description of Jacobson, cited by Husson.[1] Husson abandoned it later in favor of Goerttler's description. As we are able to show above, this latter could not suffice, even if we would accept it at its face value. *Nota bene*, any anatomical description, useful to explain Husson's idea, should establish a double set of musculature in the vocal cords, namely one for the gross movement of opening, closing and tensing ("formation") of the glottis and another for the contraction of extreme rapidity. Goerttler never attempted such a duality.

The physiology of phonation appears reduced to one question: the pitch of the voice. The ease, volume, timbre, resonance, etc. receive no further enlightenment from the neurochronaxic theory, although they appear even more important as far as our clinical considerations are concerned. (Husson himself has dedicated some thoughts to these questions and made some pertinent observations, but they were in no way derived from his neurochronaxic hypothesis.) On the whole, we could say that this new trend had impoverished voice physiology instead of enriching it.

Electrophysiology is confronted with a completely new situation. Centers of unheard-of complexity should be found, nerve conductions of never imagined frequency should be explained, muscle contractions of extreme frequency should be assumed—in short, electrophysiology would have to switch over to a completely new conception,

† On the other hand, it speaks in favor of a new trend of thought if it demonstrates its fruitfulness even before being proven definitively.

just for the sake of allowing man to sing opera.

Therapy by exercises, the mainstay of all well-considered voice therapy, loses its theoretical basis. Garde, a collaborator of Husson, noticed this quite clearly and added magnanimously that this kind of therapy "might continue but with a new connotation." What was meant by it, was never revealed. The experimentation of Garde with some drugs [42] and the hormonal studies of Amado[43] are no substitution so far for the most effective treatment, namely, that by therapeutic exercises. Voice therapy was made an orphan by Husson's hypothesis.

Wherever we look, no new explanations or solutions are offered, only an aprioristic condemnation of the established views and willful statements of revolutionary conceptions based on doubtful experimentation and unacceptable interpretations (Section IV).

If it is characteristic of fruitful hypotheses to throw more light on known facts and problems, the neurochronaxic hypothesis is certainly not of this kind. It is a simple *deus ex machina* which does not explain the old and does not prove the new.

After its rather widespread acceptance in the beginning by a number of students of the human voice (among which we seldom found a name well-established in the scientific research of phonation), lately a certain resistance and disappointment seem to prevail in the world literature concerning the neurochronaxic hypothesis. Husson can claim the credit for having electrified the field of voice research and having made it so fashionable that a great number of investigators found it interesting enough to dedicate a new effort to this field on both sides of the fence—not mentioning the daily press, which occasionally dedicated entire columns to this surprising novelty. The advantage of this resurgence of interest in voice physiology can, however, only be preserved if we continue deepening our insight along the lines of the aerodynamic theory of phonation, which has brought the science of voice to its present height and accomplishments, and abandon the neurochronaxic hypothesis which has proven to be, in order to use clinical French, a *fausse route*.

Summary

The basic experiments and assumptions of Husson's neurochronaxic hypothesis of phonation are examined as to their scientific validity; they are found inacceptable. This hypothesis, purporting that the vocal cords contract actively in the frequency of the voice and that the vocal cords "vibrate" without necessity of an airflow through the glottis, appears to be contrary to well-established facts and interpretations.

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Audiology Section

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Children with Nonorganic Hearing Problems

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Introduction

Hearing problems which are not correlated with actual pathology of the hearing mechanism have been referred to variously as "functional" losses of hearing, "psychogenic" losses, or "malingering." From the audiologist's point of view, none of these terms is completely satisfactory. "Functional" is frequently used in other contexts to refer to a loss of function which may be on an organic basis, and thus it is probably not the proper term to apply to a hearing problem which may have no organic correlate. "Psychogenic" and "malingering" refer to the presumed absence or presence of a conscious element of feigning as a part of the symptom-picture. While the psychiatrist may be able to differentiate between an unconscious variety of hearing loss, which might be considered as a conversion reaction or so-called hysterical deafness, and the conscious type of assumed loss, which is properly termed malingering, the audiologist is usually in no position to make this kind of judgment. From the audiologist's—and perhaps the otologist's—standpoint, a better term to encompass all forms of assumed hearing loss would be "nonorganic." Such a term obviously means that the pathological condition of the hearing mechanism is in doubt, and it does not attempt to identify the hearing problem as being of the conscious or the unconscious variety. As a matter of fact, to be strictly accurate, the audiologist should probably refer to nonorganic hearing *problems* rather than to nonorganic hearing *losses*, because some patients appear to have no hearing handicap except in the examination situation.

In recent years, as audiologic diagnostic methods have been refined, it has become apparent that nonorganic hearing problems are more prevalent among the adult population than had previously been assumed. Audiology centers concerned with the testing of veterans for compensation purposes are required to employ a battery of tests designed to discover the presence of nonorganic problems and to enable the examiner to determine the true organic threshold of each individual patient. In such centers, the incidence of nonorganic problems among the veteran population tested has been estimated at from 20% to 40%. Johnson, Work, and McCoy [1] have reviewed the literature and discussed the increase in the incidence of nonorganic hearing problems in military and veteran populations as familiarity with this type of difficulty has become more widespread. A nonorganic hearing problem, however, may range from a situation in which all of the patient's assumed hearing loss is nonorganic in nature to the situation in which a genuine loss exists but the patient exaggerates the true extent of his hearing problem.

As more audiologists and otologists have become involved in examining claimants in medicolegal cases, the presence of nonorganic hearing problems in nonveteran

Submitted for publication March 6, 1959.

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populations is also being discovered to a greater degree. While there are instances of such problems among adults which are not concerned with monetary compensation, these cases are relatively rare. It is commoner to find financial considerations closely tied in with the individual patient's misleading performance on certain auditory tests.

Nonorganic hearing problems may exist in children as well as in adults. In the case of children, the motivation for assuming a hearing loss is almost never associated with monetary compensation. In fact, without considerable additional study of a psychodiagnostic nature, it is not possible to establish in most cases just what motivates children to behave in the test situation as if they were hard of hearing. It behooves school audiometrists, school psychologists, teachers, administrators, and others who are concerned with the health programs in the schools to be aware of the possibility of the existence of nonorganic hearing problems in children of school age.

Over the past two years, some 40 children with significant nonorganic hearing problems have been examined at the San Francisco Hearing and Speech Center. The purpose of this paper is to discuss some findings pertaining to these 40 children and to describe the procedures which seemed to be the most effective in demonstrating true acuity.

Description of Subjects

The 40 children who constitute the subjects of this discussion ranged in age from 6 to 18 years, with a mean age of 10.9 years. Thirty-one of the children were female and nine were male. In all 40 cases the audiogram furnished by the referral source indicated a suspected marked bilateral hearing impairment when, in fact, our examination eventually revealed the hearing to be within normal limits in each ear.

Twelve children had a history suggesting previous disease of the middle ear and, perhaps, previous hearing loss of an organic nature. Fourteen were receiving special help in school, such as lipreading instruction, auditory training, and preferential seating. Two children were in special classes for the hard of hearing, and one was in a class for the deaf. One child had been furnished with a hearing aid. It should be noted that in the case of most of these children there was no indication in the referral information to suggest that the hearing loss was not as represented on the accompanying audiograms. In fact, 16 of the children were referred to the Hearing and Speech Center specifically for a hearing aid evaluation.

Most of the children seemed to be performing well academically, to be intellectually normal, and were without noticeable emotional disturbances. Nine children displayed symptoms which might be related to their "hearing problems." These symptoms included functional articulatory speech disorders, and—according to parental reports—strong sibling rivalry, persistent enuresis, anxiety reactions, possible nonorganic visual difficulties, and lack of satisfactory academic progress. It should be reemphasized, however, that children with any symptoms or history of psychological significance were definitely in the minority—less than 25% of the total group. On the basis of our own observations, we cannot state why these children performed as they did on hearing tests. We can only report how they did behave, and how we were able to establish that their hearing was normal.

Testing Procedures

It was noted that during their initial interview, 39 of the 40 children were able to follow normal conversation with no difficulty. Typically, parents reported that in their observations the children usually did not behave as if they were hard of hearing. In almost every case, poor hearing had not been suspected by the parents until this possibility had been pointed out to them by the results of pure-tone tests, usually performed in the school situation. Apparently it was only in a testing situation that all of the children demonstrated hearing problems. It was curious that only a few previous examiners had noted inconsistencies between the children's responses to conversation and their hearing test results.

At the Hearing and Speech Center, pure-tone tests routinely precede speech audiometry. In the case of these 40 children, it became apparent very quickly that their responses to the pure-tone stimuli were grossly inconsistent with their behavior in a conversational situation. Typically, these children would profess average threshold

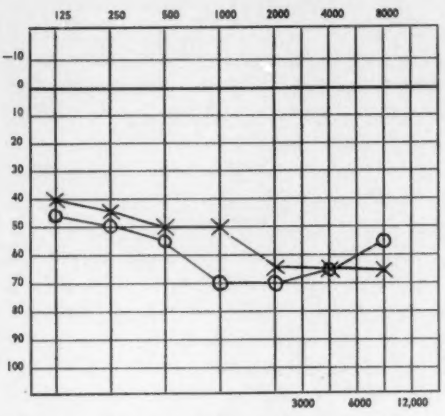

Fig. 1.—Typical initial audiogram for children with nonorganic hearing problems.

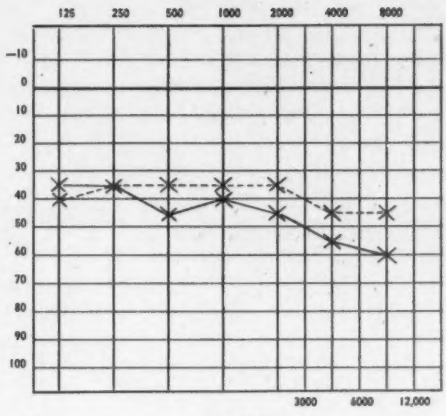

Fig. 2.—Test-retest reliability for the same ear of one child later found to have normal hearing.

levels between 40 and 60 db. at all frequencies. Their audiograms tended to be "flat" in configuration and with equal losses by air and by bone conduction. Figure 1 is a typical initial audiogram. Note that this loss pattern bears a striking resemblance to the curve of the most comfortable listening level for normal ears.

At this point, it should be mentioned that serial pure-tone audiograms, even widely spaced in time, do not necessarily provide sufficient variation to make one suspect a nonorganic hearing problem. Although we had no opportunity to estimate the ability of these children to give consistent erroneous thresholds from day to day, we were frequently provided with medical and school records indicating good test-retest reliability, sometimes over a long period of time. Figure 2 illustrates one child's ability to maintain consistent false thresholds over an interval of six weeks. The thresholds of the opposite ear showed equally small variations. This child was later found to have normal hearing in each ear.

After examining a few of these children, it became apparent that the speech reception threshold (SRT) was almost always closer to normal levels than the pure-tone thresholds. We recommend, therefore, that at the first indication one may be dealing with a nonorganic problem in a child—for example, the impression gained in the initial interview—monaural, live-voice speech audiometry be employed. The method by which one obtains the child's SRT is important. We found that best results were obtained when the SRT was approached from below, that is, by use of an ascending technique. The use of a descending or an ascending-descending method usually resulted in establishing a false SRT, and the task of measuring the true organic threshold became more difficult.

It is usually necessary to spend considerable time—and to exercise a great deal of patience—in obtaining the child's SRT. Starting at maximum attenuation, the examiner presents 6 to 10 spondee words, interspersed with such comments as, "I know it's very soft, but you must try hard to repeat these words." If the child fails to respond, the intensity of the signal is increased by 5 db. and the examiner tries again, coaxing the child to respond at almost every word. Occasionally, the examiner may insert a question, such as, "Will you take off the earphones now?" to see if any reaction is observed to indicate that the child heard the question. Also, it may be possible to engage the child in a conversation on a topic that the examiner knows is of particular interest to the child.

Thus, one child who would not repeat a spondee word at any sensation level was willing to talk about her pet dog and answer the examiner's questions when the hearing-loss dial was set at 5 db.

With the use of an ascending method and considerable urging of one kind and another, we were able to establish SRT's of 10 db. or better in each ear in 33 of the 40 children. Thus, for most of the children, it was possible to rule out any marked hearing loss by the use of speech-reception measurements. Incidentally, it was unusual to find one of these children manifesting any significant loss of speech discrimination ability. Thirty-four of the children scored 90% or better in each ear on discrimination tests administered at levels well above previously established thresholds.

In a few of the children, normal hearing could not be shown by either speech audiometry or conventional pure-tone techniques. It was then necessary to use such special tests as PGSR and delayed feedback measurements to obtain valid estimates of organic thresholds. But even in these cases, before any special testing was done, there were indications of normal hearing. These were usually found in the subject's responses to conversational voice when he was not aware that he was being tested.

At the conclusion of each examination session, after normal hearing had been established by one method or another, a final attempt was made to secure valid pure-tone thresholds, that is, pure-tone thresholds which would agree reasonably well with the speech audiometric results, or the results of special tests such as PGSR and delayed feedback. The subjects were told that the pure-tone test was being repeated because it was obvious that they had misunderstood the directions on earlier tests, or had made a mistake in judging when they should respond. Sometimes they were told that they would hear a different kind of tone which would be easier to hear than the previous ones used. Then, either a warbled tone or a pulsed tone would be employed. Valid final audiograms were obtained in the case of 33 of the children. It was possible to obtain valid thresholds with some children even when they did not respond *correctly* to the number of pulses presented. These children, after being instructed to count the number of tones they heard, would wait until a series of pulses had been presented and then blandly respond with the wrong number. But the very consistency of their responding with a number after each series of pulses, even though the intervals between presentations were widely varied, argued for the fact that they were hearing the tones. Although, as stated earlier, it is not within the audiologist's province to determine the relative consciousness of motivation in patients displaying nonorganic problems in hearing-test situations, there does seem adequate reason to believe that some of these children wilfully and deliberately attempted to deceive the examiner.

In some respects children with nonorganic hearing problems are similar in their behavior to adults with such problems, and in some respects they are dissimilar. Both children and adults tend to respond to incidental conversation in a manner that is inconsistent with their test results. Both may present a history of previous middle ear problems, but pure-tone test results that are characteristic of perceptive losses. In the test situation, however, the adult will generally exhibit some degree of loss for speech stimuli, even though the speech audiometric results may not be reasonably consistent with the pure-tone results, whereas most children will respond with valid thresholds to speech audiometry, provided the techniques suggested earlier are employed.

Summary and Conclusions

In discussing our experience at the San Francisco Hearing and Speech Center with 40 children who had nonorganic hearing problems, it has been our intention to point out that children with normal hearing, like

adults, will sometimes perform on hearing tests as if a hearing loss were present. We found this tendency to be considerably more marked in girls. Children with such problems typically do not show difficulty in understanding conversational speech. While they are likely to exhibit grossly erroneous pure-tone responses, most of them will give normal speech-reception scores when time-consuming modifications of standard speech audiometric procedures are employed. A child's consistency on serial pure-tone audiograms in itself does not rule out the possibility that he has a nonorganic hearing problem. While speech audiometry will yield valid results in most cases, in some instances it will be necessary to resort to special test procedures, such as PGSR and delayed feedback, in order to obtain valid organic thresholds. In most cases it is possible to obtain a final pure-tone audiogram which represents the child's actual hearing levels. The reasons why a child will demonstrate a nonorganic hearing problem in a test situation are obscure but apparently are not, as with adults, associated with pecuniary gain.

It is our hope that audiometrists, educators, and health authorities who deal with hearing-impaired children will be alert to observe that some children who fail school hearing tests may actually be exhibiting hearing problems of a nonorganic nature. On the basis of our observations, we recommend that children who fail school hearing tests not be placed in special classes or be regarded in any way as hearing-impaired children until the organicity of their hearing problems has been established through proper audiological study. We also suggest that nonorganic hearing problems deserve the attention of psychiatrists and psychologists in order to determine why some children behave in an aberrant fashion while having their hearing assessed.

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Single- and Double-Frequency Screening in School Children

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After studying 1,128 office patients and 1,500 school children, Glorig and House [1] concluded that in almost all cases the hearing loss at 4,000 cycles was as great as, or greater than, the loss at any lower frequency. They recommended, therefore, that testing at 4,000 cycles be used as a screening test in schools, industry, the military forces, and doctors' offices.

In a subsequent article,[2] the same authors called attention to the fact that many persons show a slight hearing loss at 4,000 cycles, something which is relatively unimportant unless accompanied by losses at the lower frequencies. They recommended, therefore, that 2,000 cycles be added in school screening programs to avoid needless referrals. That is, any child who failed to hear 15 db. at 4,000 cycles was to be tested at 2,000 cycles, and if this tone were heard at 15 db. no further testing was necessary at that time.

The advantages of either a single- or double-frequency screening test, particularly one at the higher frequencies, are obvious, assuming a fairly high degree of effectiveness in detecting hearing problems. Such screening is rapidly being accepted for use in industry and in the armed forces; considerable investigation seems necessary before it can be used with any great degree of assurance in schools. Many physicians feel that in cases of conductive deafness the low tones are affected earlier and to a greater extent than the higher ones. Since most cases of hearing loss in school children are conductive in nature, any proposed rapid-screening test should detect a very high proportion of cases of conduction deafness, even where the losses are moderate.

It was the purpose of the present study to evaluate the efficiency of the two tests proposed by Glorig and House in a group of children with known conductive deafness.

Materials and Methods

Patients consisted of 100 consecutive children 4 to 14 years of age who showed stabilized measurable conductive deafness as evidenced by a hearing loss of at least 15 db. in two or more frequencies in the same ear. All had hypertrophied adenoid tissue, a fact which is more or less irrelevant to the present investigation, and in all cases prolonged conservative therapy had failed to resolve the hearing problems. While these children do not represent a random sample of all children, or even a random sample of all children with hearing problems, they may be considered a fairly representative group of "resistant" cases which are not likely to resolve themselves. These are the cases which it is important to detect in any screening program.

All hearing studies were made by one of us (J. S.) and a skilled technician, with the use of calibrated equipment in a soundproof room in which the noise level was well below the maximum permissible for testing normal hearing. Losses were measured with a 7BP Audivox pure-tone audiometer.

Every possible effort was made to obtain hearing thresholds that represented relatively stable hearing situations rather than temporary hearing losses attributable to acute infection or allergy. In children with acute otitis media or otorrhea, audiograms were taken only after inflammation and otorrhea had subsided.

All hearing cases in this study were judged to be conductive in nature on the basis of findings in audiograms, bone-conduction tests, speech-discrimination tests (in some instances), tuning-fork tests, and physical findings.

All children eventually underwent adenoidectomy, and the number of preoperative audiograms per patient varied widely. In order to simulate a single-test screening program, only the last of the preoperative audiograms was used. At various times, a child might have somewhat better or somewhat

Submitted for publication March 17, 1959.

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worse hearing than was measured at the last visit prior to operation, but this last audiogram may be considered fairly representative of the degree of loss for each child. In 70 children, the hearing problem was bilateral; in 30, it was unilateral.

While the frequencies from 250 to 8,000 cycles had been tested, only losses at 500, 1,000, 2,000, and 4,000 cycles are included in the present study.

As a first step, a study was made of the relationship between test frequency and degree of loss. That is, do these specific children with conductive hearing losses have approximately equal difficulty on the average at all frequencies or is hearing consistently better or consistently worse at one frequency than at another?

In the unilateral cases, only the "bad" ear was used; in the bilateral cases, the loss in both ears was averaged for each test frequency. The four losses at the four frequencies were then ranked from 1 to 4 (with tied ranks for tied losses), and a Coefficient of Concordance, W[9], was computed. The Coefficient of Concordance would have a value of 1.0 if there were perfect consistency from child to child in the relative importance of the four frequencies; it would have a value of 0 if there were no consistency. In this case, W equalled 0.25 and the difference from a value of 0 was highly significant (probability less than 0.001). The means for all children were as follows: 26 db. at 500 cps, 25 db. at 1,000 cps, 20 db. at 2,000 cps, and 30 db. at 4,000 cps. Thus, with a highly significant degree of consistency, the greatest losses were observed at 4,000 cps; the smallest losses, at 2,000 cps, and intermediate losses, at 500 and at 1,000 cps. On the basis of this finding, one would expect that 4,000 cps would be the best choice of a single frequency to use in a screening program, while 2,000 cps would be the poorest choice.

One frequency at a time was used to see how many of these 100 hearing problems would have been detected with a single-frequency screening program. At 1,000, 2,000, and 4,000 cps, a loss of 15 db. or more in either ear was considered evidence of a hearing problem; at 500 cps, the critical level was taken at 20 db. because of the problem of ambient noise that might arise in the field. Results, as presented in the Table, seem to indicate that either 1,000 or 4,000 cps would be the most satisfactory single test frequency, while 2,000 cps would be the least satisfactory. At 1,000 cps there would be four false negatives and at 4,000 there would be five false negatives, all unilateral cases with rather modest losses.

Although the results in the Table seem to show little choice between 1,000 and 4,000 cps, inspection of the Figure, which gives the actual distributions of losses at each test frequency for all 100 children, indicates the superiority of 4,000 cps. It is very possible that a child with a hearing loss

*Cases "Missed" by Single-Frequency Screening Test of 100 Children**

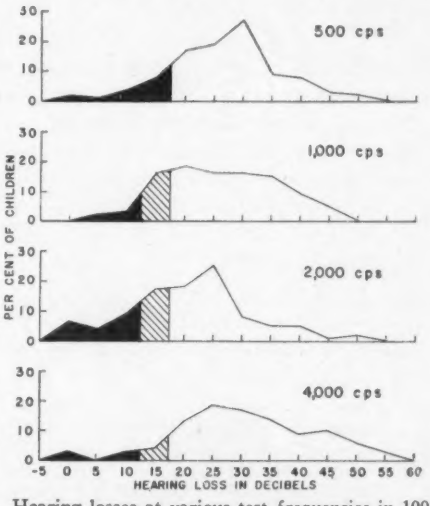
	Test Frequency, CPS			
	500	1,000	2,000	4,000
Unilateral loss	7	4	12	5
Bilateral loss	5	0	5	0
Total	12	4	17	5

* 70 with bilateral and 30 with unilateral hearing problems.

of 15 db., who was barely "caught" on a given day, would show a loss of only 10 db. on another day and be "missed." It was of interest, therefore, to compare 1,000 with 4,000 cps with use of 20 db. rather than 15 db. as a critical value. When this was done, the number of false negatives at 4,000 cps increased from five to eight, still all unilateral; the number of false negatives at 1,000 cps increased from 4 to 17, of which 13 were unilateral and 4 were bilateral. The difference between the two test frequencies was statistically significant (probability of 0.02).

When Glorig and House's second criterion was used (retesting at 2,000 cps all who "failed" 4,000 cps), 14 of the 95 children who "failed" at 4,000 cps would have "passed" at 2,000 cps. This, in addition to the five "false negatives" at 4,000 cps, means that 19 of the 100 cases would not have been detected.

If *both* 1,000 and 4,000 cps were used, all 100 hearing problems would have been detected. That is, every child in the study had a loss of 15 db. or more at 1,000 cps or 4,000 cps or both, whereas there were four "misses" with use of 1,000 cps



Hearing losses at various test frequencies in 100 children with conductive hearing problems.

alone and five "misses" with use of 4,000 cps alone. With a 20 db. criterion, there would have been 6 "misses" with both frequencies, as opposed to 17 at 1,000 cps alone and 8 at 4,000 cps alone.

Since all children included in the study had hearing problems, it was not possible to investigate fully the incidence of "false positive." There were, however, 30 "good ears," or at least there were 30 ears with only minimal problems. Of the 30, there would be 3 "false positives" at 1,000 cps and 5 at 4,000 cps (8 if both frequencies were used). While these are termed "good ears," they belong to children who are known to have hearing problems, and so the losses are probably higher than they would be in perfectly normal children. The figure quoted may therefore be taken as upper limits for the incidence of "false positives": 10% at 1,000 cps alone, 17% at 4,000 cps alone, and 27% with both 1,000 and 4,000 cps.

Comment

On the basis of these 100 children with conductive deafness, we were able to verify to a large extent Glorig and House's contention that 4,000 cps showed rather consistently the greatest hearing loss of any frequency from 500 to 4,000 cps. This phenomenon is well known in presbycusis; to some it may be a little surprising to find it in children with conductive deafness. This casts considerable doubt on the general feeling that the lower frequencies are affected first in such cases and seems to substantiate the findings of Crowe and Guild.[4]

The proposal that children be screened at 4,000 cps at a level of 15 db. seems very reasonable, since we found that only 5% of these difficult cases were "missed" with this criterion and these were all unilateral cases showing rather small losses.

While we sympathize with the effort to cut down on the number of false positives by rescreening at 2,000 cps all who had a loss of 15 db. or more at 4,000 cps, this modification seems to have some drawbacks. True, the number of false positives would be reduced, but unfortunately the number of false negatives would be increased tremendously (from 5% with 4,000 cps alone to 19%).

As an alternative test, the use of both 4,000 and 1,000 cps seems to have many advantages. Since a suspected hearing problem would be defined as a loss of 15 db. or more in either ear at *either* 4,000 or 1,000 cps, the number of false positives would doubtless be increased; the number of false negatives would, on the basis of our findings, be very small. Testing at two frequencies is of course more time consuming than testing at only a single frequency; it is still a substantial saving over testing at all frequencies.

Summary

On the basis of 100 children with conductive deafness, we were able to verify to a large extent Glorig and House's contention that 4,000 cps showed rather consistently the greatest hearing loss of any frequency from 500 to 4,000 cps.

With use of a test criterion of 15 db. at 4,000 cps, only 5 of the 100 cases would have shown false negatives.

With use of Glorig and House's proposed modification that all who failed to hear 15 db. at 4,000 cps be rescreened at 2,000 cps, 19 of the 100 cases would have shown false negatives.

With the use of *both* 4,000 and 1,000 cps, there were no false negatives in the group studied. Every child had a loss of 15 db. or more at 4,000 cps or at 1,000 cps *or* at both frequencies.

On the basis of results obtained, the use of *both* 4,000 and 1,000 cps as screening frequencies for school children seems worthy of investigation in the field.

1721 Pine St. (3).

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Case Reports

Chelation in Clinical Otosclerosis

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An improvement in hearing in a patient with nephrocalcinosis treated with edathamil disodium (disodium ethylenediaminetetraacetic acid) was reported by Clarke, Clarke, and Mosher.[1] The case was reported as hearing improvement in otosclerosis, but review of the history and nature of the hearing loss raise some question as to the correctness of the diagnosis. Regardless of this fact, it was thought worthy of repeating chelation therapy in a known case of clinical otosclerosis.

Assuming that the cause of the hearing impairment in otosclerosis is ankylosis of the footplate of the stapes to the oval window and that the pathologic progress is perivascular in location, it seems logical that if the calcium could be removed from the ankylosed stapes there would be an improvement in hearing. Sataloff [2] has recently reported that stapedes removed from fresh autopsy material specimens, when treated with a chelating agent, showed they were markedly thinned and decalcified.

The chelating agent used in this study was ethylenediaminetetraacetic acid, commonly known as edathamil.* The chelating action of edathamil has been well established.[3-6]

Report of Case

The patient is a 32-year-old white woman, a graduate student in the University. She has noted a progressive bilateral hearing loss associated with a low-pitched tinnitus of about 10 months' duration. Her mother has had a similar hearing complaint for about 30 years and uses a hearing aid successfully. Physical examination was entirely within normal limits, except audiometric studies which showed a bilateral conductive type hearing loss averaging 35 db. (left) and 27 db. (right) in the speech frequencies by air conduction and normal bone conduction. Blood calcium and phosphorus levels prior to treatment with edathamil were 9.5 mg/100 ml. and 3.4 mg/100 ml., respectively, determined by the method of Carr and Frank.[6] These chemistry studies were repeated biweekly during chelation and showed no change.

Daily intravenous administration of 3 gm. of edathamil disodium in 500 cc. of 5% dextrose was given over a three- to four-hour period for five days, followed by two days without. This cycle was repeated three times (i.e., until edathamil disodium had been given for a total of 15 days). Daily urinalysis was done, watching particularly for albumin, renal casts, and renal parenchymal cells.[7] All urinalyses were normal. Pyridoxine was available to counteract any side-effects, such as nausea, diarrhea, abdominal cramps, and skin reactions, which have been noted.[6] A syringe of calcium gluconate was always available at the bedside to prevent any hypocalcemic tetany, which is a theoretical possibility. Neither pyridoxine nor calcium gluconate was ever necessary.

Weekly audiometric examinations were done during chelation which showed no improvement in hearing. Very slight improvement which has persisted was noted on the fourth-month postchelation test (Figs. 1 and 2).

The patient had, on Dec. 22, 1958 (one year post chelation), a left stapes mobilization operation with very satisfactory results (Fig. 2). It is not known whether the previous chelation therapy aided in the ease of mobilization. The hearing in the right ear (not operated on) has remained the same (Fig. 1).

Conclusion

A single case is presented of use of edathamil disodium in early otosclerosis. Minimal improvement in hearing was

Submitted for publication Feb. 15, 1959.

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* Furnished by Dr. Rodney Gwinn, of the Abbott Research Laboratories, North Chicago, Ill.

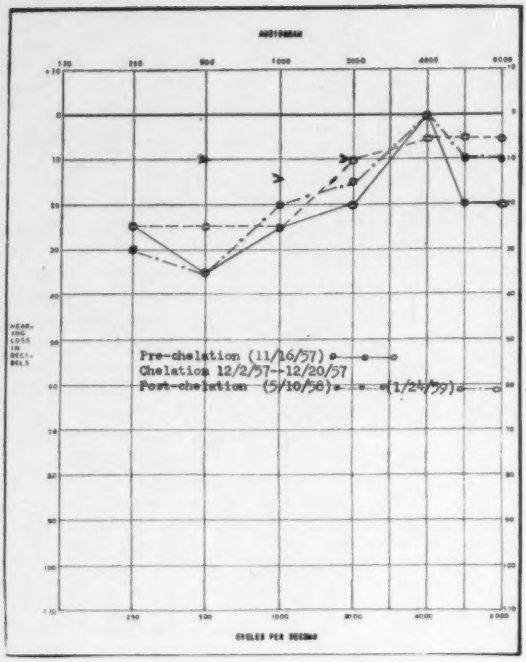

Fig. 1.—Pure-tone (AC and BC) audiogram of hearing responses in right ear. Solid line indicates before chelation; broken fine, four months post chelation; dot-dash line, one year post chelation.

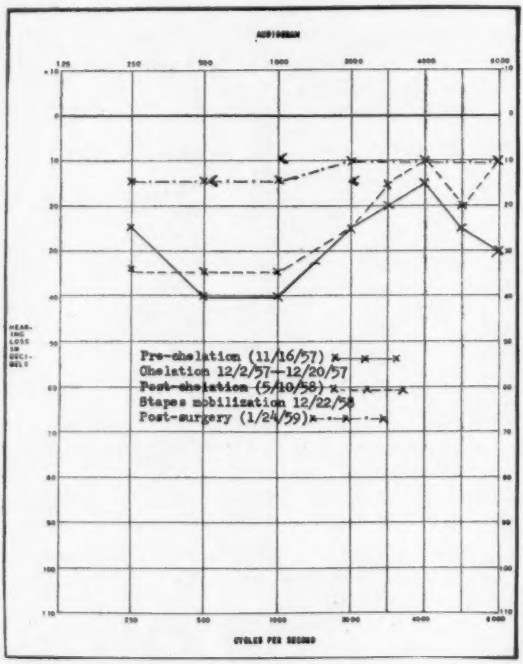

Fig. 2.—Pure-tone (AC and BC) audiogram of hearing responses in left ear. Solid line indicates before chelation; broken line, four months post chelation; dot-dash line, one month post stapes mobilization and 1 year post chelation.

noted. Further research should be conducted before any conclusions can be made on the clinical usefulness of edathamil disodium in otosclerosis.

Department of Otolaryngology, University of Wisconsin Medical School (Dr. Bennett).

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The Use of Free Skin Grafts for Post-Traumatic Cicatricial Stenosis of the Laryngopharynx

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The use of free skin grafts for reconstruction of the pharynx after laryngopharyngectomy for neoplastic disease is well documented.[1,2,5,8-16] However, the opportunity to use these grafts for benign post-traumatic and postinfectious cicatricial stenosis of the pharynx finds infrequent application. A review of the literature reveals very few recorded instances of this use.[3,4,6,13]

The mechanism producing post-traumatic stenosis must be extraordinary. Many of these patients probably die as a result of the initial injury and never reach the stage of stricture formation. We recently have had experience in managing one such case who lived to develop a stricture and merits recording.

Submitted for publication March 23, 1959.

Presented before the Otolaryngological Staff Meeting at Texas Children's Hospital, Jan. 13, 1958.

From the Department of Otolaryngology, Baylor University College of Medicine and Affiliated Hospitals.

Report of Case

The 8-year-old son of circus performers sustained an injury to his neck and pharynx when he was pulled by a rope about his neck for a distance of 300 ft. by a runaway horse. The injury included fracture of three cervical vertebrae without neurological sequelae and hematoma of neck and hypopharynx that required tracheotomy.

He was first seen by us three months after his injury, at which time he had a well-developed stricture of his hypopharynx and was wearing a cervical brace. At this time, the patient could only eat soft foods and liquids.

Artist's drawings are shown in Figure 1(*a*) and (*b*), indicating the location and size of the stricture. Radiographically, it appeared as in Figures 2 and 3.

Under general anesthesia, the stricture was incised in two areas and the patient fed per Levin tube for four days. However, in spite of

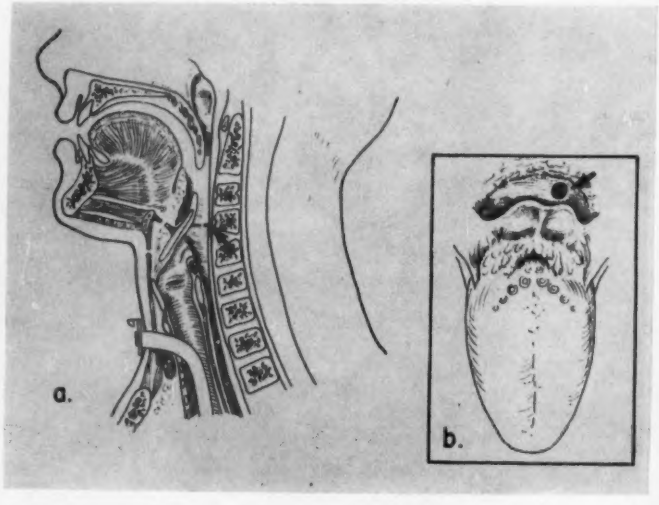

Fig. 1.—(*a*) A lateral-view drawing, showing the approximate area of the stricture. (*b*) An artist's version of the appearance of the stricture by indirect, or mirror, laryngoscopy.

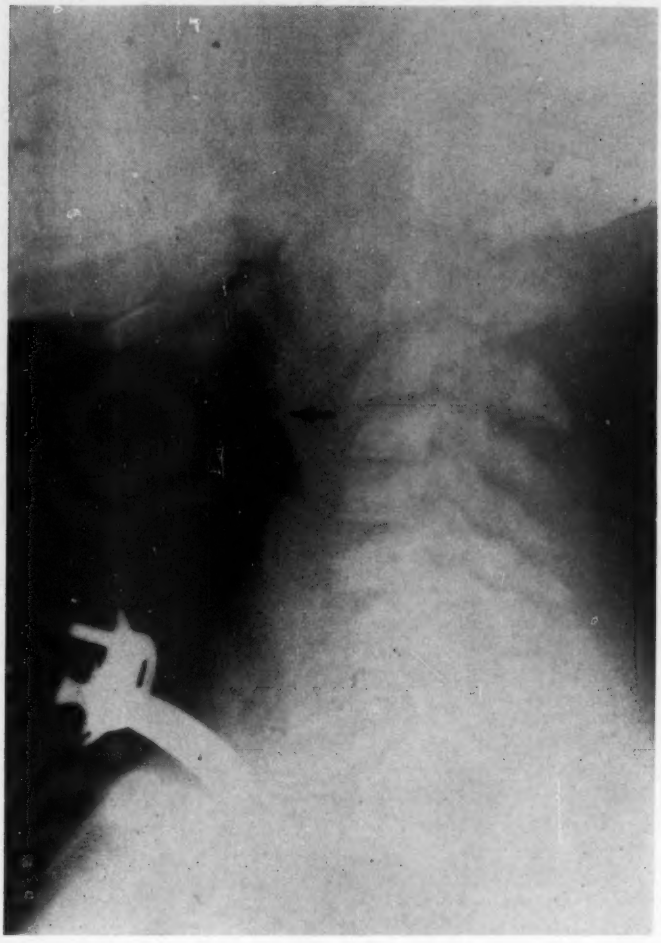

Fig. 2.—A lateral view of the neck, showing the stricture at bast of epiglottis.

bougienage with a Hurst mercury bougie, the stricture recurred.

Therefore, three months after the above operative procedure the patient was taken to the operating room, where, under general anesthesia, the stricture was incised in multiple areas (Fig. 4*a*). A free skin graft approximately 18/1,000 in. thick was taken from the thigh, wrapped around a mold fashioned from a finger cot filled with sea sponge, and placed in the region of the incised areas. The stent was then fixed in place with transfixion suture through the neck (Fig. 4*b* and *c*).

Postoperatively, the patient was fed per Levin tube for 13 days. Eleven days postoperatively, the mold was removed. On laryngoscopic examination, it was apparent that the graft had taken in all areas.

Three months after this operation the patient underwent fusion of the cervical spine without incident.

The patient is now seven months postoperative, and there is no appreciable recurrence of the stricture. The patient eats all types of food and has no respiratory difficulty. The appearance of the hypopharynx is as in Figure 5.

Comment

Free skin grafts find good application in the oral cavity and pharynx. We have been using these for a number of years in patients with pharyngolaryngectomy.

This is our first experience with a benign situation, and it points out the futility of

Fig. 3.—The stricture when contract media is used with a relatively normal pharynx above and a relatively normal laryngopharynx below.

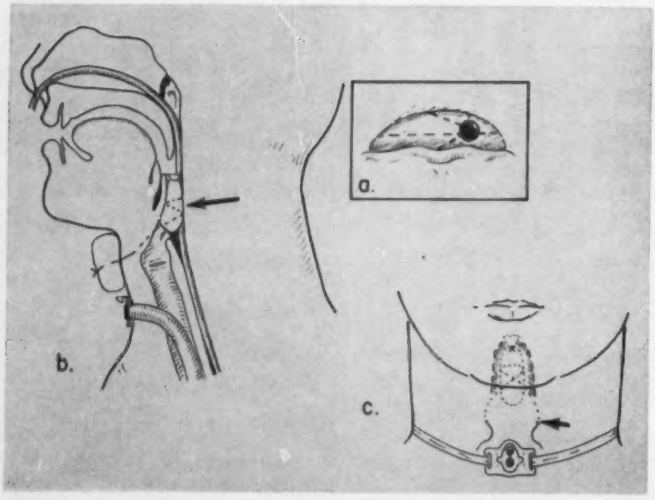

Fig. 4.—(*a*) Approximate lines of incision of the web or stricture. (*b*) and (*c*) The position of the suture which was passed through the tissues of the web below the hyoid bone and wrapped around the skin-covered stent to hold it in place.

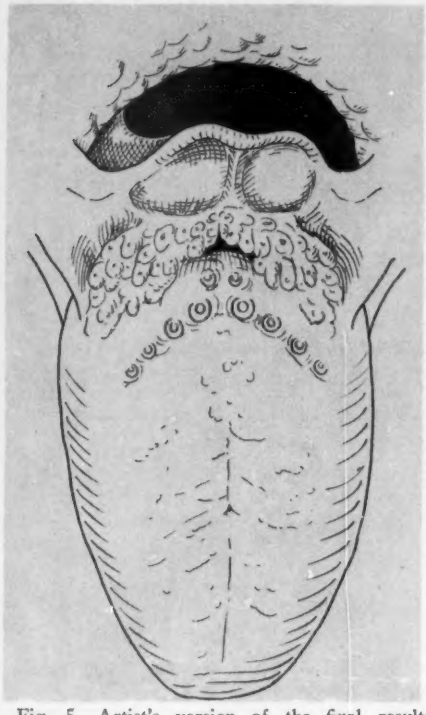

Fig. 5.—Artist's version of the final results (six months postoperative) as viewed by indirect laryngoscopy.

simple incision and dilatation when skin grafts can be used. Important factors to be considered are the thickness of the graft, application of graft to the surface to be covered, suitability of the recipient site, and immobilization of the site to be grafted. Grafts that are too thick have less chance of taking, and those that are too thin may be easily macerated by the mold or stent. We use grafts 15/1,000 to 20/1,000 in. in thickness.

Good application of the graft to the recipient site is of the utmost importance. In the cervical esophagus and hypopharynx after laryngopharyngectomy, we have been using synthetic aortic grafts with corrugated walls as a stent with good results. For intralaryngeal grafting we have used finger cots filled with sea sponge as stents and have been pleased with the application of the graft to the recipient site.

One must obtain immobilization and reduce contamination. Introduction of the nasogastric tube through the stent and transfixion through the neck has satisfied these criteria in all applicable instances. Transfixion is usually not necessary after laryngopharyngectomy.

We believe that the foregoing case report, with satisfactory result, is exemplary of the good results that can be obtained in utilization of free skin grafts to the hypopharynx, provided certain criteria are fulfilled.

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Progress in Otolaryngology

Plastic Surgery

Summaries of Bibliographic Material Available for 1957

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Cause and Prevention of Cleft Palate and Other Congenital Deformities

Efforts to prevent cleft palate and other deformities have been hindered in the past by the assumption that most congenital defects are determined by a chance arrangement of genes and chromosomes. This has served to direct the efforts of research workers to other areas where the possibilities for success have seemed more promising.

The results of a number of extensive studies regarding the cause of deformities in animals, however, indicate that maternal disease and other critical stresses occurring at certain stages during pregnancy may be responsible for a number of congenital abnormalities including cleft lip and cleft palate.

Of particular interest are the studies made on pregnant mice and pregnant rats. In the mouse and rat acquired abnormalities have been produced in the offspring by subjecting the pregnant animal to a number of treatments with various agencies, all of which are capable of producing congenital deformity.

The stress factor is defined rather broadly and includes oxygen deficiency, exposure to x-ray, vitamin deficiency, cortisone intoxication, and virus infection. As examples of stresses inducing deformity in animals, Warkany has produced eye defects and cleft palate by rendering the pregnant animal vitamin-deficient. Baxter and Fraser have induced a high incidence of cleft-palate deformity in the offspring of pregnant mice by giving them cortisone. Ingalls produced cleft palate and other deformities by rendering the pregnant animal anoxemic or oxygen-deficient.

The human undoubtedly has a hereditary tendency to produce occasional cleft-palate offspring. Statistically, this is expressed by the incidence of harelip or cleft-palate deformity or both occurring in about one instance in 1,000 births. Approximately 25% of these families will have a history of similar deformities on the paternal or maternal side, going back as far as grandparents. In about 75% of the cases there is a negative family history for harelip or cleft-palate deformity.

A reasonable assumption would seem to be that, in the 75% of patients with a negative family history, two factors may be required to result in cleft-palate or cleft-lip offspring—the genetic racial tendency for cleft-palate or lip deformity strengthened or reinforced by a *stress factor* during the mother's pregnancy. The stress factor, to be effective, must be exerted at the particular time during pregnancy when the palatal processes are undergoing their rapid period of development, which in humans is near the end of the ninth week of pregnancy.

Measures to Prevent Cleft Palate

In our laboratory * we repeated the work of Baxter and Fraser; we induced 85% occurrence of cleft palate in offspring by subjecting pregnant mice to injections of

Submitted for publication March 31, 1959.

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cortisone acetate between the 11th and 15th days of pregnancy. We then conducted a series of experiments in which individual B-vitamins were given concomitantly with the cortisone acetate to neutralize or render ineffective its teratologic effect. Of 10 vitamins tested, the administration of only pyridoxine (vitamin B_6) and folic acid resulted in lowering the incidence of cleft-palate deformity in the offspring. Consequently, we are routinely administering pyridoxine and folic acid during a subsequent pregnancy to patients who have had a child with cleft lip and palate. Thus far, all of the offspring of these mothers have been normal, but the series is not large enough to be significant.

Homotransplantation of Tissues

Important breakthroughs in the homograft problem to date are as follows:

1. It is now generally accepted that skin and other tissues can be exchanged between identical twins with the expectation that these transplants will behave like grafts of the patient's own tissue (autografts).

2. Patients with agammaglobulinemia (a condition in which these persons lack the capacity to form antibodies) will accept grafts from other persons as they would autografts. There is also evidence that patients with uremia and those in terminal stages of cancer will likewise tolerate homografts for longer periods of time than normal persons. These conditions probably represent deficiency states in which the person's antibody formation against the antigen of the graft has become greatly depressed.

3. Great expectations have been predicted as regards a person's tolerance to skin and other tissues from stillborn infants (embryonal grafts). Surface skin embryonal grafts, which can be accurately followed-up, appear to enjoy a longer survival time when transplanted to an adult. The longest-surviving embryonal skin grafts in humans, however, have not exceeded 40 days. Buried embryonal grafts, such as endocrine glands, are reported to survive for longer periods of time, but this survival in most instances is based on the assumed physiological activity of the transplants rather than from biopsy studies. The whole matter of embryonal tissue grafts as homotransplants requires further investigation.

4. In our laboratory, full-thickness skin grafts have been exchanged between parents and infants when the latter were being operated upon for closure of harelip defects. The parent, whether mother or father, was chosen on the basis of blood compatibility as to the A, B, O, and Rh factors with the infants. In 65 skin exchanges between parents and their children, a definite pattern of homograft behavior was established as follows: All grafts exchanged between father and infant were rejected, the longest survival being 14 days. When the skin grafts were exchanged between mother and offspring, however, 25% of the grafts were tolerated by both mother and child.

5. Children who tolerate their mother's skin may also tolerate other tissues from their mother, such as cartilage, endocrine gland, and kidney. Routine skin exchanges between mothers and children, therefore, may prove to be useful tests for possible tolerance to other tissues exchanged between mother and child.

Skull and Scalp

Longacre and deStefano [1] report on the successful use of 60 split rib grafts in 11 patients, with extensive defects of the cranium and facial skeleton. Not only have losses of 40% to 60% of the cranium been repaired by this means, but also traumatic defects of the frontal, supraorbital, and malar bones. Split rib grafts have also been used to rebuild the extensive bony deformity in severe hemiatrophy after new blood supply had been furnished by means of a jump flap under the atrophic skin. The mandible has been reconstructed and the retruded middle third of the face secondary

to cleft palate repair has been corrected by such bone grafts. In three cases in which previous severe infection had occurred and the refrigerated bone grafts had to be removed before the infection would subside, there was no loss or destruction of the split rib graft due to infection, extrusion, or absorption.

Splitting the rib opens up the cancellous trabeculae and thus insures the survival of a high percentage of osteocytes, which will envelop the graft with new bone very rapidly. The split rib graft possesses enough strength to protect and, at the same time, serve as a scaffold to carry these cells.

Within 35 days there will occur the regeneration of fine new ribs of the same length though slightly thicker, and such regenerated ribs have been found to possess excellent osteogenic properties.

In the opinion of Lewis, Blocker, and Eade,[2] extensive defects of the scalp, either alone or associated with underlying loss of bone, may be the result of congenital deformities, vascular lesions, benign and malignant tumors, radionecrosis, infection, and all types of trauma, including burns. In the surgical approach to these lesions, the peculiar anatomy of the scalp must be taken into account. In the scalp, three layers of tissue are so firmly bound together that they function as one fairly inelastic covering, with the skin fixed to the subcutaneous tissue and the blood vessels fixed to it. Circulation is so abundant that ready healing occurs in lacerations which would be held to be impossible or precarious to close elsewhere. Nevertheless, there is the danger of communicating infection to the intracranial venous channels.

To avoid either exposure of the brain or the denuded cranial bone, immediate coverage must be obtained at the time of trauma or surgery by skin grafts, or preferably by flaps from the adjacent areas taken with the base toward the periphery of the scalp. In lesions of the forehead and frontal area, skin grafts placed directly on the pericranium will be under great tension and subject to cicatricial shortening and breakdown; they should be replaced by flaps as soon as practicable. Over the occipitofrontalis or the aponeurosis, skin grafts do well. The authors consider preserved cartilage and autogenous bone the choice materials for replacement of loss of bone in skull defects of suitable size; they prefer tantalum plate for very large defects where adequate soft tissue is present.

Whalen[3] compares two cases of avulsion of the scalp to show the advances that have been made in plastic surgery. Treatment demands an immediate cover to obtain a closed wound and limit complications. Thus far, this is best achieved by the split skin graft, and it is not possible to obtain satisfactory permanent hair growing from replaced avulsed scalp tissue.

In the first case, further loss of the scalp occurred due to infection. Small deep grafts were ineffective, and the defect was allowed to heal by scar epithelium. During 50 years the scalp has never remained fully healed.

In the second case, shortly after an accident, abdominal split skin grafts were placed over the entire defect, with an almost 100% take. A large flap of hair-bearing skin from the posterior remaining scalp and upper neck was fashioned, delayed, and rotated anteriorly to the forehead. The stages of forming, delaying, and finally advancing the flap is diagrammatically illustrated.

The patient is able to dress her hair. There has been no psychic upset.

As pointed out by Moss,[4] premature synostosis of the frontal suture occurs three times more frequently in the skulls of cleft-palate patients than in the skulls of those without cleft palate. Marked ridging of the ectocranial surface over the site of the suture indicates the presence of such a synostosis. The incidence of such ridging is greater in males than in females. Premature frontal synostosis and ridging are associated with malformation of the skull base.

Moss hypothesizes that the basal malformation induces changes in the organization of the dura mater; this is related to the synostosis. Such a malformation in cleft palate patients is correlated with the high incidence of frontal synostosis in this group.

Face

In immediate treatment of traumatic injuries of the face, Owens [5] emphasizes the absolute necessity of gentle handling of the tissues throughout. Tissues should be handled with small skin hooks or small plain forceps. Complete hemostasis must be achieved by means of the finest pointed hemostats. The first consideration in a case of craniofacial trauma is the presence of an adequate airway. Hemorrhage must be controlled. Damage to the facial nerve is to be kept in mind as a probability. To appraise the examiner of the status of the nerve, the patient should be asked to raise his eyebrows, to pucker his eyes tightly, to wrinkle his nose, to bare his teeth and whistle, and to tighten his platysma. The status of the teeth is most important.

In treatment, thorough cleansing of the wound is of first importance. When tissue must be excised, the excision should be planned to follow Langer's lines of tension on the skin.

To fulfill the basic requirements for dealing with tumors of the lip, ear, or eyelid borders, O'Connor, McGregor, and Safier [6] have used a block "V" excision. The parallel lines remove a block of tissue with the immediate lymphatics included. When the block "V" incision is converted into a straight suture line, immediate additional length is obtained to prevent retraction as well as to effect a smooth closure distally. Proper contour of the part and a fine linear scar are the usual end-result. This surgical procedure has served the authors well in many facial problems when tissue has to be revised, removed, or transplanted.

A case report is presented by Edwards [7] in which bilateral temporal neurotomy was carried out for frontalis hypermotility associated with face lifting. The forehead of the patient had a fleeting "concerned" look during normal conversation. The temporal incisions, made in face lifting, were connected across the scalp at the hairline and the skin elevated. An incision was made at right angles to the course of the temporal branch and carried down to bone. After this bilateral division of the temporal branches of the facial nerves the forehead skin was drawn up and trimmed in the usual manner. The patient is very pleased with the result.

Grooves in the skin of the forehead and drooping eyebrows were corrected by Barnes [8] by incising the skin above the brow and resecting the corrugator supercilii along its entire extent. The forehead was undermined up to the scalp hairline. The frontalis attached to the skin of the forehead was cross hatched. The pyramidalis nasi was avulsed with the hemostats. All vessels along the incision line were controlled by temporary clamping. Numbness may last several months.

Dowd [9] reviews various plastic procedures to reconstruct defects of the head and neck after excision of malignant lesions. These procedures include (1) direct closure, (2) extensive undermining with direct closure, (3) the use of local flaps, (4) the use of split-thickness skin grafts, (5) the use of full-thickness skin grafts, (6) the use of distant flaps, (7) the use of composite grafts, and (8) metallic reconstruction of the mandible after radical resection of the neck and jaw.

Dowd emphasizes complete removal of all diseased tissue and adequate reconstruction of defects of involved regions, such as the nose, eyelids, ear, scalp, cheek, lip and tongue, so that the patient may return to his normal place in society.

Zovickian and Cooper [10] report a case of osteoma of the frontal sinus which was removed, and the frontal defect was replaced immediately. The tumor was exposed by removal of the inner table of the

sinus and was delivered en masse. Cancellous bone chips from the iliac crest were used to fill the frontal sinus cavity, and the outer table of the frontal bone was replaced.

Skin

In selected cases, Greeley and Curtin [11] revise skin grafting techniques with better results than formerly. They inspect the graft site early, delay grafting when oozing of blood and serum is expected, and consider the use of wet dressings as having a good effect on the graft. Longer delay in skin grafting is encouraged after excision of radiation lesions and in areas where fibrosis has extended through the deep fascia and periosteum.

Calnan and Innes [12] became very doubtful of the effectiveness of pressure as a factor in the "take" of a skin graft. They experimented with application of split skin grafts on a suitable raw surface without any dressing, with favorable effect. The late results made it clear that pressure on grafts is not necessary for their successful take. Calnan and Innes investigated the effect of cooling skin in refrigeration at 3 C to 6 C before application in various areas, which resulted in complete take.

They arrived at the following conclusions:

Cooling of grafts allows them to survive in vitro for a length of time that is roughly inversely proportional to the temperature, within physiological limits, i.e., the lower the temperature, the longer the time of survival.

Exposure of skin grafts applied to the body so that they are at room temperature appears to prolong their survival during the time of the "plasmatic circulation."

The exposure of grafts is practicable and appears to offer certain advantages clinically.

During a period of two years, Broadbent and Bailey [13] made a bacteriologic study of refrigerated skin grafts in cases, without special selection, in which skin grafting was required. One hundred samples of skin were cultured, specimens being examined twice at three-week intervals; thirty-eight were from surgically clean cases, and sixty-two samples were from surgically dirty cases. On initial culture, 18 (36.8%) of the clean and 34 (54.8%) of the dirty cases were positive. Good "takes" of the grafts were obtained in all cases except three, and these were "dirty" cases in which Proteus organisms were combined with nonhemolytic Staphylococcus pyogenes var. aureus. Broadbent and Bailey concluded that bacterial contamination by itself is not the usual cause of graft failure.

The principle of the method for treatment of scars presented by Hynes [14] is to remove the surface of the scar by shaving and then to obtain rapid healing of the raw area by covering it with a skin graft. If the surface of a matured scar is removed by shaving, the raw surface of the fibrous tissue exposed shows a multitude of tiny, freely bleeding points. A skin graft "takes" most readily when it is placed on a defect with a free capillary circulation. If, therefore, a skin graft is placed on a shaved scar, it takes rapidly and only a light dressing is required.

A large number of thin unstable scars on the limbs allow the removal of a very thin surface layer by shaving, showing the usual multitude of bleeding points. A thick skin graft will take on the free capillary circulation. Thus the use of distant flaps can be avoided.

Good results are obtained with mildly contracted and tight scars, with depressed scars, and with scars which are difficult or even dangerous to excise. Permanently red or brown scars can also be treated in the same way, and the results appear very promising.

Four cases of keloid scarring following incisions on the back of the ear for correction of congenital deformities are described by Patterson.[15] Tension due to insufficient removal of cartilage or excessive removal of skin and prolongation of the postauricular incision over the top of the ear are possible

etiological factors. In the four cases presented, there was a long latent period following initial operation before typical keloid scars developed. The keloid scars were excised.

Though extensive pigmented nevi are now commonly treated by shaving off the superficial layers, as pointed out by Patterson,[16] Morrant Baker, in 1873, experimentally shaved off a small piece of the surface of a hairy mole on the forehead of a girl. The result was so encouraging that the whole area was given the same treatment. When healing was complete, one month later, an absence of contraction was noted. He gradually removed the regrown hairs by application of nitric acid. The final scar, after disappearance of the mole, contained tiny patches of pigmented skin here and there with a very few fine hairs.

Ear

The problem of congenital external auditory canal atresia is discussed by Anthony,[17] and a bilateral case is reported. The left ear had a deformed and fused malleus and incus (which were removed) and a bony tympanic membrane. A Thiersch graft was taken from the right thigh, a portion was placed in the floor of the cavity, and strips were sutured to the skin edges of the orifice (Woodman's procedure). In the right ear, the mastoid was unpneumatized and the stapes was found to be congenitally absent. According to Anthony, only two other operations revealing the congenital absence of the stapes have been reported.

Four patients with acquired complete external auditory canal atresia, in two of which previous corrective surgery had not been satisfactory, are presented.

It would seem to Anthony that the technique used for the acquired atresia may be relied upon to produce satisfactory results. Additionally, it would appear that the prognosis is adversely affected and surgery probably contraindicated in the unpneumatized mastoid cases of congenital atresia.

Prosthetic devices for the prevention of stenosis were not required in any of these patients. Instead, a half-moon of cartilage was removed from the concha, immediately posterior to the endaural incision after undermining the skin, thus creating a small flap which was packed in place posteriorly. Splitting the upper end of the endaural incisions postoperatively was not necessary to prevent stenosis, because in most cases primary or secondary skin grafting of these surfaces was done.

McCash[18] inserts into the helix of a reconstructed ear fine polyethylene tubing threaded over a stainless-steel wire. This procedure has been found effective in preventing soft tissue contraction from occurring between the successive stages of the reconstructive procedure. The two ends of the wire are twisted together in a loop. There have been no complications in the five cases in which this method has been used. Where a cervical tube pedicle is employed around the rim, it is of particular value in keeping it expanded during the contractile phase. McCash suggests that the method could be used in stabilizing the donor ear after removal of the maternal ear cartilage.

To increase the area of a patch of skin and subcutaneous tissue, Neumann[19] buried an inflatable rubber balloon, the gradual extension of which caused sufficient expansion of the skin of the side of the head to provide covering for a cartilaginous graft during the repair of a subtotally avulsed ear. A flexible polyethylene tube, through which the balloon could be inflated, was passed from the balloon through a subcutaneous tunnel inferiorly down the side of the neck to emerge through a stab wound. After three weeks, the balloon was gradually distended by the introduction of successive small increments of air, which was prevented from escaping by a stop-cock on the exposed portion of the polyethylene tube. After two months, the skin under tension had expanded approximately 50%. At the second operation, the balloon was

removed through a postauricular incision, and a fresh autogenous C-shaped costal cartilage graft was introduced. The expanded skin was fashioned about the graft as a double-pedicled tube flap. Later, a series of small multiple "Z" flaps were employed to blend the inferior pedicle of the reconstructed portion of the ear with the superior portion of the residual ear.

Eye

As viewed by Sherman,[20] unquestionably, there is no better source of skin for reconstruction of the upper eyelid than the opposite upper eyelid, if available, and posterior auricular skin is the second choice. He has found the method of Hughes' lower eyelid reconstruction a very satisfactory procedure when applied to the upper lid, especially when not much more than the tarsal portion is lost. He would also prefer to use it when practically all the lid is missing.

Dowd[21] discusses common lesions of the eyelids and orbit, such as hemangioma; ptosis of the upper lid; epithelial inclusion cysts; lacerations and fractures of the maxilla involving the infraorbital ridge, the floor of the orbit, and the malar bone; thermal injuries, and diplopia due to fracture. Contracted sockets requiring skin grafts are best treated with split-thickness grafts. After removal of a wax stent, an acrylic mold, fastened indirectly to a denture by Stader splint bars and bolts, is used temporarily. Extensive malignant lesions of the lids require repair of the resulting defect by the application of full-thickness skin grafts. If the greater portion of the lid is involved, Hughes conjunctival-tarsal flap is the procedure of choice for repair.

As viewed by McCoy,[22] management of facial trauma is usually made difficult by deformities of the orbit or dysfunction of the eye. In fracture of the orbit, proper position and function of the eye and its associated structures are primary objectives. The commonest deformity is downward and backward displacement of the globe as well as diplopia in one or more spheres.

In superior orbital fractures without intracranial problems, replacement of bone fragments with stabilization is sufficient. For reducing lateral fractures, McCoy prefers the Gillies approach, introducing a No. 21 cervical sound beneath the temporal fascia and exerting upward pressure against the depressed fragment. Reestablishment of the floor of the orbit at the time of the repositioning of the orbital ring in inferior fractures is best accomplished by open reduction with direct wiring of the fragments. In recent medial fractures frequently attended by extensive comminution in the nasomaxillary area, forcible reduction is best carried out through two angular incisions just anterior to the inner canthi. McCoy uses direct wiring methods, Kirschner wire pinning, and even the plaster head cap when indicated.

It is the belief of Converse and Smith[23] that traumatic enophthalamos is due to an enlargement of the orbital cavity; the orbital fat distributed in a larger cavity no longer suffices to maintain the normal protrusion of the eyeball. Diplopia occurs in comminuted fractures of the orbital floor with downward displacement because the visual axis of the affected eyeball is not in alignment with that of the opposite eye.

Converse and Smith advocate early bone grafting at the time of the accident. In all such fractures, they favor a wide intraorbital exposure of the maxilla and zygoma by an incision made in the mucosa on the labial aspect of the sulcus (Converse, 1950). Injury to the infraorbital neurovascular bundle is avoided. If the floor is fractured and depressed, a more satisfactory exposure is obtained by an incision through the skin of the lower eyelid, orbicularis oculi muscle, and periosteum of the rim of the orbit. Reduction of the fractured zygoma is achieved by leverage through the temporal or oral approaches, and reduction of the maxillary fragments is done under direct vision through the intraoral exposure. Re-

alignment of orbital rim fragments is best obtained through the lower lid incision. An autogenous bone graft from the ilium restores the continuity of the floor and its proper level. Illustrative cases are presented.

Smith [24] reviews some of the mechanisms of diplopia in fracture of the orbit. Diplopia, being a subjective condition, is a symptom to which introspective persons and malingerers are inclined. The results from tests for diplopia must therefore be correlated with objective findings. Occlusion, the red glass test, and other tests are evaluated.

Three types of diplopia are differentiated: physiological, monocular, and binocular. The reasons for the occurrence of diplopia are explained. Smith feels that all depressed fractures of the orbital floor should be surgically reduced and maintained by a mechanical means. The best method of treatment is direct exposure of the fracture through a skin incision. Large fragments of a comminuted orbital rim may be drilled and wired, and numerous small fragments should be supported on the surface of an autogenous iliac bone graft.

Diplopia remaining after all bone work has been completed should be treated conservatively until further improvement fails to take place. In terminal muscle surgery, an attempt should be made to move the field of single binocular vision into a central position.

In a new operation described by Schimek,[25] a strip of orbicularis muscle, which holds the lower portion of the tarsus in position, is tightened. This prevents inversion of the upper border of the tarsal plate. Incisions, preferably horizontal, are made 3 mm. below the lid border and temporal and slightly above the outer canthus. A 0000 black nonabsorbable surgical (silk) suture is tied about a bundle of orbicularis in the lid incision. Both arms of the suture, threaded on a long intestinal needle, are passed subcutaneously over to and out of the temporal incision. One arm of the suture, threaded on a curved needle, is passed thru the orbicularis and fascia in the temporal incision. The suture is tightly tied so that the lid border is everted, cut, and allowed to remain buried. The skin incisions are closed with interrupted sutures. In this procedure there is a minimum of dissection and bleeding. A temporary ectropion must be produced to guard against any recurrence of the entropion. This procedure is particularly of value for recurrent senile spastic entropion with scarring from previous cautery or surgery.

Nose

Marcks, Trevaskis, and Payne [26] present a method to elongate the columella by transferring two single pedicle flaps from the outer borders of the prolabium and by rotation and interdigitation in the form of a Z-plasty. The procedure is well illustrated in diagrammatic drawings. The base of the flaps is within the nostril floor. Both flaps incorporate the scar tissue and a portion of the prolabium in order to add more width. In some patients the interdigitation of just one flap is sufficient, especially if the prolabium is off balance or if the one side is badly scarred.

Huffman and Lierle [27] present a well-illustrated and soundly written article on progress in septal surgery. They classify the surgical procedures directed at the anterior part of the septal cartilage into four main categories: (1) subtotal removal, (2) total resection and replacement as a graft or grafts, (3) mobilization and reposition as part of a compound flap, and (4) provision of pliability by surgical weakening.

Any particular type of procedure discussed by the authors (or any combination of them) may be needed for correction of any individual septum. The surgeon should be able to handle all of them with facility but with the knowledge that any of them may produce changes in the external nose demanding immediate or delayed repair. The concept of treating anterior septal deflections with minimal removal of skeletal elements appears worthy of consideration.

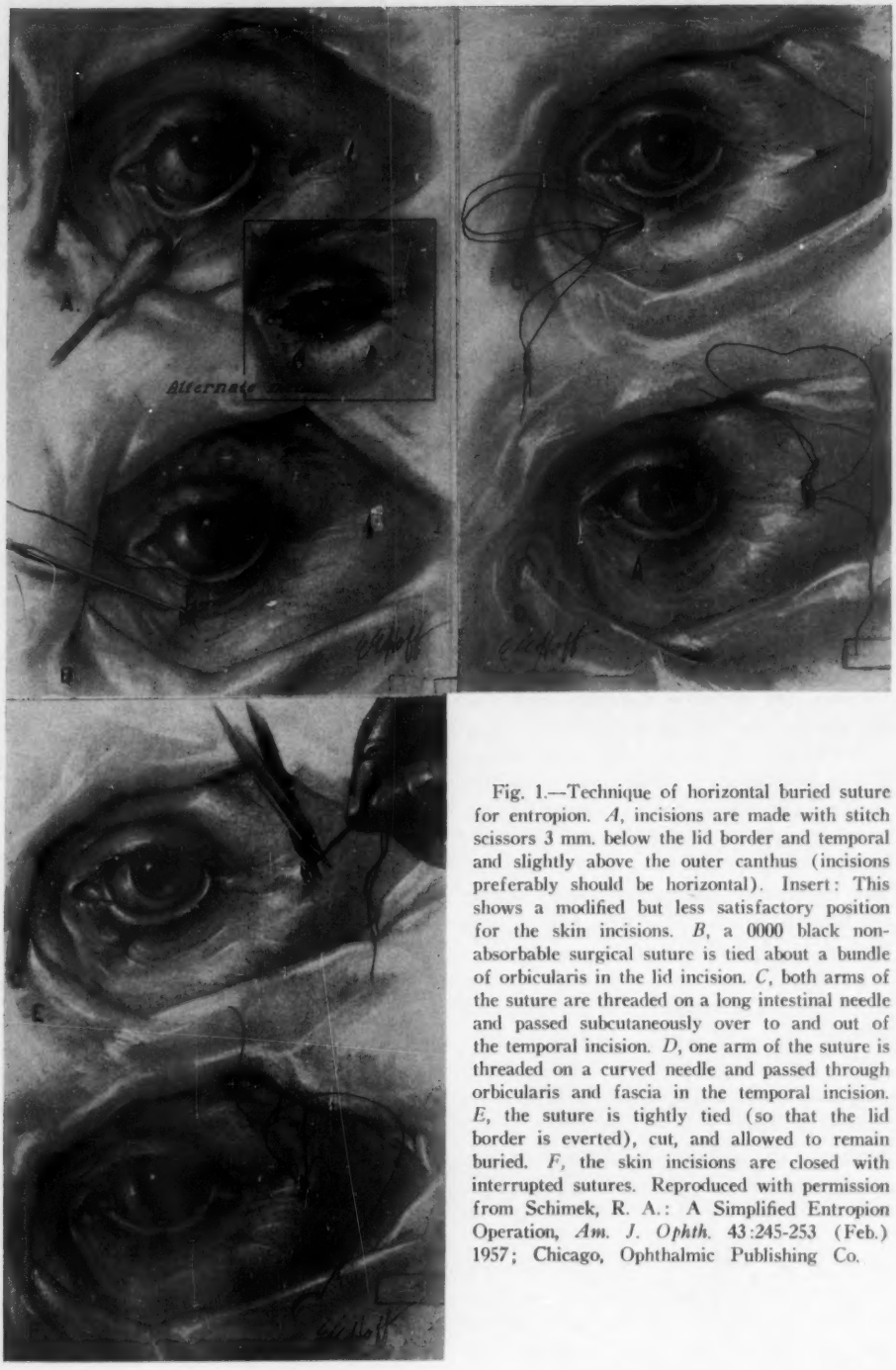


Fig. 1.—Technique of horizontal buried suture for entropion. *A*, incisions are made with stitch scissors 3 mm. below the lid border and temporal and slightly above the outer canthus (incisions preferably should be horizontal). Insert: This shows a modified but less satisfactory position for the skin incisions. *B*, a 0000 black non-absorbable surgical suture is tied about a bundle of orbicularis in the lid incision. *C*, both arms of the suture are threaded on a long intestinal needle and passed subcutaneously over to and out of the temporal incision. *D*, one arm of the suture is threaded on a curved needle and passed through orbicularis and fascia in the temporal incision. *E*, the suture is tightly tied (so that the lid border is everted), cut, and allowed to remain buried. *F*, the skin incisions are closed with interrupted sutures. Reproduced with permission from Schimek, R. A.: A Simplified Entropion Operation, *Am. J. Ophth.* 43:245-253 (Feb.) 1957; Chicago, Ophthalmic Publishing Co.

As viewed by Wynn,[28] the nasolabial flap in the face is attended with little danger; the length of the flap can be three times longer than the width of its base because of the rich vascular supply in this location. It is important to determine where suture lines of the incisions will fall after the flap is rotated. The incisions heal well within the crease produced by the nasolabial fold and often are barely visible after four to five months. Careful attention should be given to hemostasis before the flap is sutured into position. The flap should always be trimmed and the surrounding tissue adjusted by undermining in order that the flap can be sutured into position without tension. Complications and the method for handling them are described. Wynn considers the nasolabial flap an excellent means of reconstructing nasal defects in the nasal glabellar area, lateral nasal wall, and nasal ala, with a minimal postoperative cosmetic deformity.

Straith, Straith, and Lawson[29] add to their former presentation (i.e., a method to improve the one-sided flattened nasal tip and ala and the wide flat nostril remaining after repair of a lip cleft). Repair of nostril deformity is carried out secondary to repair of the lip. A Z-plasty in two planes is used, one flap on the skin side of the nasal tip and the other on the mucous membrane side. In the present article, the authors stress the application of this method on both sides of the nasal tip in the reconstruction of double harelip noses. The double Z-plasty elongates the columella without additional visible scars to the nose or the lip and is combined with a cartilage graft or cartilage flap for support.

In their method of reconstruction of the syphilitic saddle nose McLaren and Penney[30] divide the operation into three stages: (1) dissection and excision of scar tissue, (2) preparation of the mold for carrying the graft, and (3) application of the graft. In edentulous patients, the upper Gunning type splint is wired into place in the first stage. The alar cartilages and soft tissues of the nose are freed from the bony rim of the pyriform opening. The soft tissues are lifted forward off the underlying maxilla into an exaggerated position of the normal. To correct the tip-tilting, several incisions are made within the nose into the raw subcutaneous surface down to the entire length of the dorsum. The "flaring" of the nostrils can be corrected by vertical "darts" cut into the skin of the inner opening of each nostril vestibule.

An impression of the cavity is then taken with black Stent composition, large enough to stretch the skin. A broad sheet of thin split skin taken from the arm or thigh is wrapped around the mold and introduced through the oronasal opening into the cavity. This is followed by firm fixation of the mold and graft. An acrylic mold can normally be introduced in place of the original composition mold two weeks postoperatively. The mold is later replaced by the final intranasal prosthesis.

In the seven cases reported, good results were obtained.

Wilson[31] uses a tripartite incision in the surgical approach to the nasopharynx (as described by him in 1951) in treatment of choanal atresia. The article is diagrammatically illustrated.

The incision is made through the whole thickness of mucous membrane down to the bone, and flaps are separated—two laterally and one posteriorly. The posterior flap is dissected backward to the posterior border of the bone, and the nasopharynx is opened by dividing the mucosa transversely along the edge of the bone. The incision can be extended laterally and backwards on the affected side for as far as is necessary to give an adequate view of the choana. The mucous membrane of the posterior border of the septum should be divided and the mucous membrane separated from front and back of the atresia and from the unaffected side of the posterior part of the septum. Mucous membrane flaps are formed to cover the raw areas. The soft palate is now brought forward and the

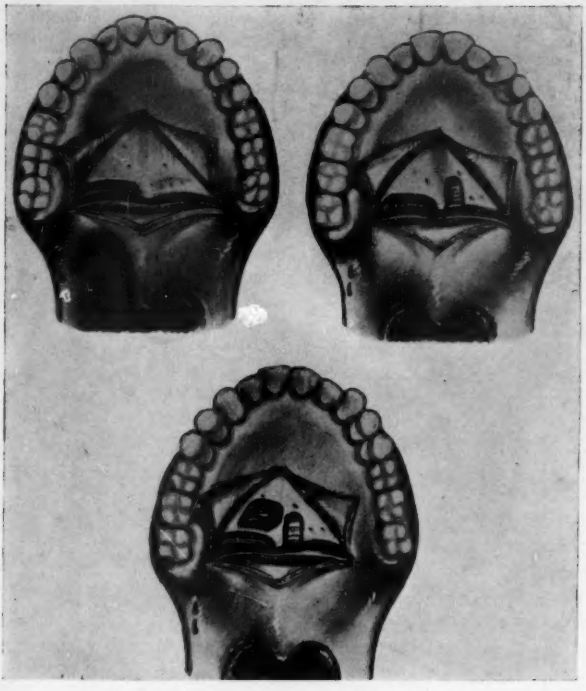


Fig. 2.—Progressive stages in removal of bone of hard palate. Upper left, posterior edge of hard palate removed up to the atresic area. Upper right, juxtaseptal portion of bone removed from hard palate on unaffected side. Lower, bone removed from hard palate on affected side as far backwards as the area of atresia. Reproduced with permission from Wilson, C. P.: Treatment of Choanal Atresia, *J. Laryng. & Otol.* 71: 616-625 (Sept.) 1957; London, Headley Brothers.

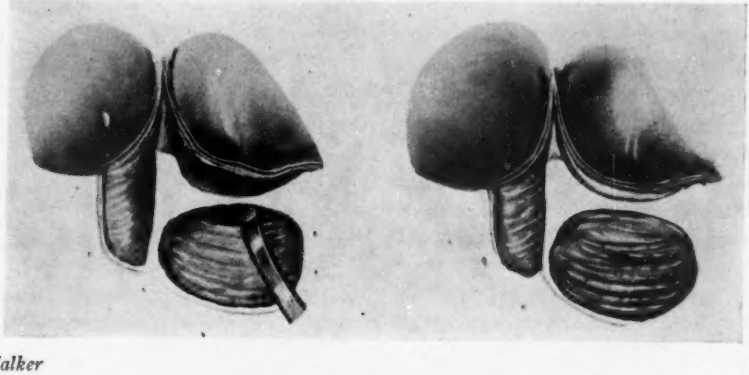
mucosa of its upper surface sutured to the new posterior border of the floor of the nose on the affected side.

Jaw

The cases reported by Stuteville [32] demonstrate the reconstruction of the mandible in patients with deformity due to congenital defects, to destruction of the growth centers by trauma, and to surgical removal of a portion of the mandible.

The problem of replacing lost growth centers caused by congenital absence or traumatic destruction of the heads of the mandible in the early years of growth is interesting and new. In animal experi-

Fig. 3.—Left, impression of Figure 2 Lower, viewed from behind, to show choanae, with division of the mucosa of the posterior border of the nasal septum. Right, the mucous membrane has been separated from the bone of the posterior part of the septum and the front and back of the atresia. Reproduced with permission from Wilson, C. P.: Treatment of Choanal Atresia, *J. Laryng. & Otol.* 71:616-625 (Sept.) 1957; London, Headley Brothers.



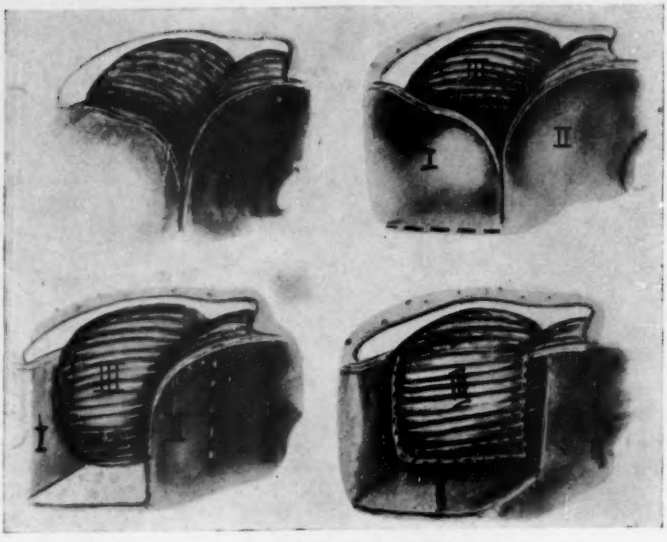


Fig. 4.—Upper left, the bone of the atresia has been removed and also the posterior part of the septum. Upper right, the three areas of mucous membrane which will form the flaps to cover the raw areas. Dotted line shows the incision in Flap I. Lower left, Flap I has been laid in position and dotted lines represent incision in Flap II. Lower right, Flap II has been laid in position and lines of incision in Flap III are shown. Reproduced with permission from Wilson, C. P.: Treatment of Choanal Atresia, *J. Laryng. & Otol.* 71:616-625 (Sept.) 1957; London, Headley Brothers.

ments, various growth centers have been transplanted into the area of the head of the mandible and a continuation of growth obtained. However, since this is a new procedure, Stuteville feels that the clinical use of this method should not be taken lightly.

In case studies, the use of the costochondral growth center of the rib, of the metatarsal growth center of the foot, of cartilage from costal arch, and of bone from the ilium have been presented. In two cases of tumor in which removal of the mandible was necessary, reconstruction was carried out immediately during the same operative procedure.

For total loss of the mandible, Erich[33] combines the use of a tubed flap of skin and an intraoral prosthetic device. This technique involves the preparation of a clavicular tube flap of skin, which is elevated and eventually inlaid in a horizontal direction between the skin and the floor of the mouth. In this technique, Erich has attempted to transform the ridge of scar tissue between the floor of the mouth and the lower lip and cheeks into a sort of alveolar ridge over which the prosthesis is ultimately placed. More satisfactory results have been effected by this technique than by any other technique previously employed.

Whether definitive treatment for maxillofacial injuries in children be instituted immediately, as stated by Georgiade and Pickrell,[34] depends on the exclusion of other associated injuries. The usual wiring techniques used for older patients are often not suitable when deciduous teeth must be wired. Fractures of the maxillary area in children are most frequently due to a forward fall.

An appliance or procedure should be kept as simple as possible in order to restore proper occlusion and function. There should be a minimal interference with the deciduous and underlying permanent tooth follicles. Elbow restraints may be necessary to prevent dislodgement of the previously applied oral appliances. Precautions should be taken to allow proper feeding and administration of fluids, regardless of the type of appliance used.

In prognathism, Erich[35] favors the removal of a segment of bone from either side of the body of the mandible which divides the lower jaw into three fragments. In the first operative stage, teeth in each segment of bone to be removed are ex-

tracted and two saw cuts are made through the alveolar process on each side, followed by fixation of the teeth. After three or four weeks, the second and third stages may be carried out. In the second stage, a segment of bone is removed from one side of the mandible without injuring the nerve, and in the third stage, a segment of bone is removed from the opposite side. The anterior fragment is then moved back into apposition with the posterior fragments, and they are held together with absorbable (gut) or nonabsorbable (tantalum wire) surgical sutures. Then, intermaxillary rubber bands are applied, bringing the teeth into occlusion and immobilizing the fragments. In Erich's experience, this three-stage procedure has been more satisfactory for the correction of mandibular prognathism than any other method which he has employed.

As reported by Lewars [36] in his series of children, many of the fracture lines of the horizontal ramus of the mandible were found to be relatively more widespread than in adults. Compared with the commoner fractures seen in civilian adults involving one or two teeth, fracture lines in these children involved from 2 to 14 teeth and a correspondingly greater proportion of bone. The statement that in children all jaw fractures involving teeth are compound fractures may not be so because, in the early stages of development, unerupted teeth may be involved while they are still deeply situated in the bone. Their presence or absence does not hasten or retard union of the fracture. Rapid union may occur in comminuted simple fractures in children. Injury to a crypt does not necessarily cause serious injury to the tooth it contains. Fracture of the horizontal ramus can cause malocclusion either through loss of teeth from the fracture line or through deformity of the bone resulting from malunion.

As stated by Harding,[37] in patients subjected to cancer surgery in which a partial resection of the mandible is necessary, it is important to maintain stability of the mandible and pharyngeal funnel after operation. He suggests a method, in selected cases, of preparing a devital anatomical bone splint by salvaging the resected bone, cleaning out the diseased portion, and then sterilizing the residual bone in the autoclave. The splint is then fixed to the retained segments of the mandible. It has the proper shape and strength for replantation and stability, provided enough normal bone remains in the specimen to give mechanical support. The evidence seems to indicate that the autoclaved bone acts as a foreign body, becomes surrounded by a fibrous capsule, and is resorbed slowly over a long period of years.

A technique was developed by Georgiade, Altany, and Pickrell,[38] experimentally, on animals, to keep bones from growing together, by means of autogenous dermal grafts. In the procedure on eight patients with ankylosis of the temporomandibular joint area, an autogenous dermal graft was interposed between the newly cut and contoured bone edges of the condyle, with excellent functional and cosmetic results. In one incidence of partial recurrence of the ankylosis in which a bilateral ankylosis procedure had been performed at the same time, without fixation of the dermal graft, correction was carried out in a second procedure. The closer to the anatomical temporomandibular joint the operative procedure is performed, the better one can expect the mandibular movements to be. A meniscus-like tissue with a resultant new physiologic mandibular joint is produced.

Lip and Mouth

Hyslop [39] advocates early debridement of electric burns of the lips and angles of the mouth. The importance of getting these patients into the hands of a reconstructive surgeon without delay is stressed. The natural line of demarcation between the devitalized and normal is a fleeting one; it being in evidence for a period of only about 12 hours. By following along the plane of demarcation with a blunt pair of

scissors, one can easily remove the tissue involved in the burn without sacrificing normal tissue. In connection with the debridement it is necessary to extend the dissection beyond the red areola of the burn in order to remove all thrombosed veins insofar as possible. Hyslop applies the same manner of debridement to electric burns elsewhere on the body with gratifying results.

Comment.—In our hands we have found this early method of treatment to be very advantageous.

According to Crikelair and Hickey,[40] "whistle deformity" not infrequently follows the repair of a complete or incomplete bilateral prealveolar cleft in which the lateral segments of the lip are sutured to the prolabium, retaining the vermilion of the prolabium as the center segment of the lip.

Among the possibilities of repair, a more satisfactory procedure would seem to Crikelair and Hickey to be a V-Y type of advancement in one operation. A flap of mucous membrane along the submucosa is advanced downward and outward while based at the mucocutaneous line. Incisions almost at the mucocutaneous line, at the juncture of the central deficient segment with the full lateral components of the lip, are carried backward and upward around the free edge of the lip, forming a triangular flap bilaterally. Each incision is carried almost to the apex of the buccal-labial sulcus, forming two larger triangles. The flap thus formed is then advanced into position so that the central segment is equal in fullness to the lateral portions of the lip. After the small triangular flaps are thus advanced forward, triangular wedges of mucous membrane of similar size are removed from the lateral segments of the lip. The entire flap is then sutured into its new position. Drawings illustrate the technique.

This procedure has also been of value in occasional unilateral lip repair and in electrical deformities resulting from burns of the lip with similar vermilion deficiency.

Paletta, Coldwater, and Booth[41] believe that excision of the entire vermilion area of the lip is the best treatment for extensive leukoplakia and that a V-excision of the lip should be combined with "lip shave" in the presence of demonstrated carcinoma. Carcinoma in situ of the lower lip can be mistaken for leukoplakia. Total excision of the vermilion and vermilion border is advised because of the tendency of leukoplakia to give rise to multicentric carcinoma. The authors prefer surgery to irradiation in the treatment of lesions of the lower lip associated with leukoplakia.

Study of their cases indicates that carcinogenic activity is confined to the mucous membrane, since multiple microscopic sections frequently reveal foci of carcinoma, and new lesions often develop in the remaining vermilion when a "lip shave" has not been done.

As long as ineptly performed repair of oral cavity malignancy and residual disease occur, as pointed out by Edgerton and Desprez,[42] for an unnecessary number of patients with such a lesion, many physicians and laymen turn to x-ray or radium therapy.

In a well-illustrated article, they present the reconstructive technique used in the plastic service at The Johns Hopkins Hospital on 154 patients undergoing surgical treatment for cancer of the oral cavity. Although the majority of these patients (69%) were failures after irradiation, a three-year survival without evidence of recurrence was obtained in 43% of the cases, which included lesions of the cheek, floor of the mouth, tongue, alveolus, palate, tonsillar fossa, and hypopharynx.

The importance of combining reconstructive methods immediately with excision of the primary cancer and regional lymphatics is stressed.

Cleft Lip and Cleft Palate

The principle of conservation of the skin vermilion ridge employed in secondary cleft-lip repair is now applied to bilateral

cleft-lip repair by Marcks, Trevaskis, and Payne.[43] They definitely feel that the skin-vermilion ridge of the prolabium belongs in with the skin vermilion ridge of the rest of the lip. A few suggestions regarding the prolabium are therefore made.

1. Preserve it in its entirety.
2. The length or width is no factor whatsoever. It will increase in size in all dimensions.
3. Never place skin beneath the prolabium initially.
4. Preserve the entire ridge—place it from the vertical to the horizontal and avoid a tight, flat lip. If there is a tight lip, the lower lip will be redundant. In direct proportion, if there is a relaxed upper lip, the redundancy of the lower lip will not be too evident.

Stenström,[44] of the Swedish Hospital in Pusan, Korea, uses LeMesurier's technique for bilateral as well as unilateral harelip repair. Both sides are operated on in the same stage. A wedge-formed excision was made on both sides of the midline of the prolabium for receiving the quadrilateral flaps from the lateral segments, and the lateral cut lines of the excisions were made somewhat shorter than the medial ones. In primary cases, the prolabium was made more movable by dissecting it from the premaxilla.

Representative cases of primary repair of complete and incomplete cleft lip and of secondary repair are reported. Good lip eversion was always obtained, and usually the lip was readily movable. The results, evaluated only a short time postoperatively, are considered as good as those of any other method.

Broadbent[45] removes the total lip surface to remove the scars indicative of a cleft lip and "cat whisker" scars. He describes a procedure for total resurfacing of the badly scarred bilateral cleft lip with a free full-thickness clavicular graft. The procedure is illustrated in a case report.

Surgery of the double cleft lip and protruding maxilla as evolved in his hands is described by Cronin.[46] The prolabium is used to form the full vertical length of the middle of the lip. When this is done, an esthetically pleasing lip is obtained. The vermilion is left attached to the prolabium inferiorly, and its thickness is increased by crosslapping of a lateral vermilion flap beneath it. Six to eight weeks before the lip repair, excessive protrusion of the premaxilla is corrected by excising a rectangle of vomer and accurate Kirschner wire fixation of the fragments. In this manner, bony union is obtained.

From a review of approximately 750 cases of cleft lip-palate in reference to dental problems at the Indiana University Medical Center, Trusler and associates[47] found that the factors are congenital maldevelopment, surgical trauma, and dental caries. In many cases, all three of these factors contribute to the problem, and frequently it may be difficult to assign the principal causative factor. That in severe deformities there exists the possibility of increasing the morbidity by ill-advised trauma is pointed out. The authors believe that many of their patients should have orthodontia directed toward correction of malformations in the maxillary bones very early in life, long before the permanent teeth have erupted.

Cronin[48] believes that the push-back operation for lengthening the short cleft palate and the congenitally insufficient palate rests on sound principle. As is commonly carried out, there is one major defect in the operation, namely, the large raw area on the nasal surface of the soft palate. As this area heals, there is shortening, sometimes crucial in amount, because of scar contracture. Split skin grafts have been used by some surgeons to cover the area, but this requires a two-stage procedure.

Cronin describes and illustrates his technique, with the use of flaps of nasal mucosa. Flaps outlined by incisions of the mucoperiosteum are elevated back to the posterior bony margin. Flaps of mucosa obtained from the floor of the nose are used

to cover the area ordinarily left raw on the lengthened soft palate. The raw area is thus shifted from soft tissue, which is subject to contracture, to a bony surface resistant to contracture.

Longacre and deStefano[49] performed the posterior pharyngeal flap procedure with and without retropositioning of the palate on 52 patients in various conditions. In follow-up, they noted a gradual change occurring in the old badly scarred cleft palates. These became increasingly vascular owing to new blood supply obtained from the posterior pharyngeal flap. In most of the patients, sufficient length of the palate was obtained by turning down a hinged flap from the nasal side of the soft palate and anchoring the posterior flap with its pedicle from above into the denuded area. In others, it was necessary to do a repositioning of the entire palate. In a few patients, extraoral tissue had to be used.

As pointed out by Metzger,[50] as a result of recent studies, the deformity of cleft lip and palate must be regarded as a craniofacial anomaly in which aberrations of bone growth may be as important as the palatal deficiency. It is thought that the causal factors are multiple, and attention is primarily turned to developmental faults in the arterial system. Older surgical procedures could fulfill only rarely the requirements of adequate velopharyngeal closure. The use of the Wardill-Veau-Kilner operation is suggested. Flaps are developed in the manner of a V-Y plasty and allow for almost indefinite increase in palatal length. A review of the causes of speech disability would indicate that the surgical intervention can be most valuable when applied before poor speech habits are developed.

Metzger discusses the advantage of mechanical and surgical obturation.

Salivary Glands

Disease of the major salivary glands, according to Lathrop,[51] is not uncommon. In the majority of instances, this is due either to a disturbance in the development of the function of the gland or to its involvement by calculus formation, inflammation, or neoplastic process. Occasionally, it may be the result of tumors arising from the nerves, blood vessels, or lymphatics contained in the gland, or, in rare instances, it may be a manifestation of an obscure condition of unknown origin. In addition, a salivary fistula and paralysis of the face may be the residual of a facial laceration which involved a salivary gland.

Calculi, occurring most commonly in the submaxillary gland or its duct, should be removed surgically through an intraoral or an external approach, but when recurrent or multiple calculi affect the submaxillary gland, excision of the gland is the treatment of choice. Acute inflammations occur most frequently in the parotid gland and usually respond satisfactorily to adequate hydration and antibiotic therapy.

In a report by Byars, Ackerman, and Peacock,[52] 23 (5%) of a series of 470 salivary gland tumors occurred in patients below the age of 18. Fourteen of seventeen mixed tumors were situated in the parotid gland, one in the submaxillary gland, and two were removed from the palate. All of these tumors were excised, and there were no postoperative nerve palsies. Of the 10 patients with mixed tumor of the parotid and submaxillary gland who were followed up, five are well after 10 years and two have been followed up over 5 years with no recurrence. There were seven tumors that did not arise from salivary gland tissue, but were found within the substance of the parotid gland.

Six tumors (26%) were malignant and showed a predilection for white females. Pain and rapid growth were characteristics of malignant tumors not found in benign lesions. Facial nerve palsy was not present in any children with cancer. The principles of management of salivary gland tumors and the operative technique are presented.

To treat salivary gland tumors, Gaisford and Hanna[53] stress the imperative

need of a thorough knowledge of the pathology. They present methods of diagnosis of the various tumors and point out that, with moderate experience, one can usually differentiate infections, cysts, and tumors. A surgical technique in approach and removal of parotid tumors is described. Though surgery is considered the best treatment in numerous instances, it is necessary to resort to x-ray therapy. Benign and malignant salivary gland tumors, which are not uncommon among children, must be promptly and adequately explored surgically and a proper form of therapy carried out. It must be borne in mind that tumors of these glands may occur at a distance from the major salivary glands and may be treated surgically. The fear of causing a facial paralysis should not be a deterrent to the performing of adequate surgery.

As pointed out by Clifford, Kelly, and Brush,[54] carcinoma of the salivary glands is uncommon and rarely presents the classic signs of cancer for clinical diagnosis. They advise that the diagnosis and plan of treatment are best made at the operating table. The treatment of the disease is only moderately successful. All painful lumps in the neck, especially in the middle-aged patient, must be viewed with a high degree of suspicion. The patient must be prepared not only for the technical procedure but must be made aware of the possibility of permanent facial disability.

Esophagus and Pharynx

Hamacher[55] presents two cases of nasopharyngeal stenosis as a complication of infection following difficult adenotonsillectomy, in which surgical reestablishment of a normal airway resulted in improved phonation. He points out that phonation is also improved by surgical creation of nasopharyngeal stenosis in instances of velopharyngeal incompetence.

In Hamacher's procedure in two cases, a set of flaps are elevated from the oral surface of the soft palate and retracted forward, the base being inferiorly placed on the posterior pharyngeal wall. A second set of flaps are constructed from the posterior pharyngeal wall and superior half of the stenotic band, based on the soft palate side and undermined and released from the posterior pharyngeal wall. These flaps are then brought forward and sutured into the defect created by the first set of flaps on the oral side of the soft palate. The first set of flaps are then sutured into the defect on the posterior pharyngeal wall created by the second set of flaps.

Walden et al.[56] present a tracheotomy anesthesia technique in surgical treatment of the pharyngeal flap. On the basis of their experience with 53 cases in which tracheotomy anesthesia technique was used, they believe that this method has definite advantages. These are, facility of the method when performed on a small pharynx, a less encumbered working area, better control of anesthesia, facilitated preparation of the flaps and control of bleeding, increased ease in performing the operation, decreased operative time, and no problems of postoperative bleeding and edema.

The development of a carcinoma in a prethoracic skin tube is reported by Jayes.[57] At the time of operation, the tumor involved a small segment of the esophagus above the anastomosis, but its main spread was down the skin tube, with very little involvement of the surrounding tissues. After esophagodermatojejunogastrostomy, the patient had lived a normal life, with practically no difficulty in swallowing, for 21 years. Then a fistula over the manubrium sterni developed. The involved portion of the esophagus was removed, together with a wide margin of surrounding tissue and overlying skin. The upper end of the original esophagus was brought to the surface in the left side of the neck, and the divided end of the skin tube was left open at the level of the fourth intercostal space. The defect on the chest wall was closed by local rotation flaps, and rapid healing occurred. Later, complete reconstruction was carried

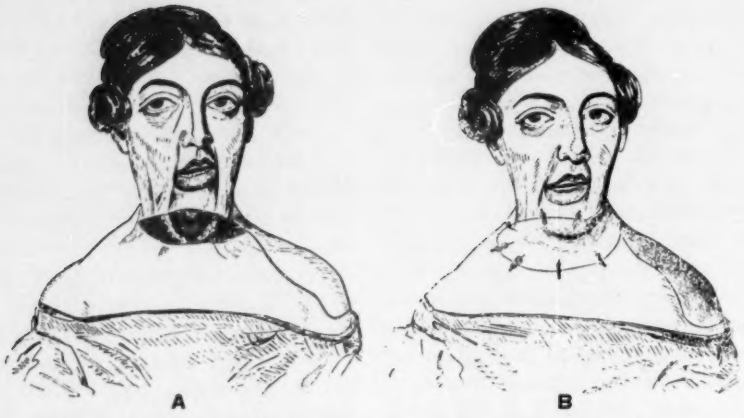


Fig. 5.—Illustrations from Prof. Thomas Mütter's paper, published in 1842, showing his original method of correcting cicatricial contractures of the neck with an "autoplasty technique" utilizing adjacent tissue from the shoulder. Reproduced with permission from Zovickian, A.: Pharyngeal Fistulas, *Plast. & Reconstruct. Surg.* 19:355-372 (May) 1957; Baltimore, The Williams and Wilkins Company.

out in one stage with the use of designed local flaps.

Large shoulder flaps based superiorly on the mastoid and occipital regions are employed by Zovickian [58] to repair pharyngeal fistulas occurring in radical surgery or radiation for cancer. He does not claim the procedure of shoulder flaps to be original and gives credit to Thomas Mütter (1842), who used it in the treatment of severe contractures of the neck due to burns. Later, Staige Davis employed this procedure in the same manner. As far as can be determined, this type of repair has not been adapted to the prevention of salivary fistulas of the neck.

The flap is prepared in two operative steps 7 to 10 days apart. Successful transfer of the flap is carried out 14 to 21 days after the two preparatory operations.

Cases are reported and illustrated to demonstrate the reliability and versatility of this type of flap. Its further advantage is that it furnished coverage with a permanent blood-bearing pedicle.

As explained by Calnan,[59] Passavant's ridge is a localized forward projection of the posterior lateral pharyngeal walls which fades out anteriorly. This ridge is too low, too slow, too inconsistent, and too easily fatigued to be of essential importance in the normal speech mechanism. It is suggested by Calnan that "Passavant's ridge" should be mentioned only in small print, as a footnote, in works dealing with speech, for its interest as the basis of another medical myth.

Parotid Gland

A method of staining the parotid gland in the live animal is described by Forrest and Robinson.[60] A No. 22 polyethylene tube, having been inserted into the dilated orifice of Stensen's duct, is held steady as 4 cc. of sterile 1% aqueous methylene blue is injected. This staining method delineates only parotid tissue with patent intact ductal system. In nine patients with parotid tumors, no difficulty was experienced in totally staining the parotid gland. These patients were treated by parotidectomy after preliminary staining of the gland.

As viewed by Stuteville,[61] there are three classes of disease of the parotid gland that may require surgical intervention: inflammatory diseases, tumor, and trauma. Treatment ranges from simple drainage to wide block dissection, including radical neck

dissection. Four types of inflammatory complications requiring surgical treatment are described: complications of mumps, obstruction of the parotid duct by calculi or inflammation, acute parotitis, and secondary involvement of the parotid gland in tuberculosis.

The discussion of tumors of the parotid gland includes mixed cell tumors, both benign and malignant as well as mucoepidermoid tumors, and other benign tumors in the region of the parotid gland. The mucoepidermoid tumor is separated from the general classification.

Stuteville discusses injuries to the parotid region, including injuries to the gland itself and to Stensen's duct.

A case report of a child in whom the hemodynamics of a cavernous hemangioma of the parotid gland were particularly evident is presented by Backus and DeFelice.[62] The patient could make the mass appear quickly on request by "holding his breath." In surgical excision with preservation of the facial nerve, the mass was exposed through a preauricular incision with a low curving cervical addition. The main trunk of the facial nerve was readily isolated, and the ramifications overlying the mass were dissected and preserved as the hemangioma was completely excised.

A study of the literature dealing with end-results in the treatment of mixed tumors of the parotid gland and 65 cases taken from the files of The Johns Hopkins Hospital were correlated by Winsten and Ward.[63] It is evident that the conservative methods of therapy for these tumors are inadequate for permanent cure. Even a cure rate of 90% should not be acceptable, and most surgeons do not obtain even this result. The highest cure rate results from total parotidectomy.

The high recurrence rate (30.0%) after supposedly adequate surgery is confirmed in this study. The incidence of permanent accidental facial paralyses showed a direct relationship to the extent of dissection.

Early operation for mixed tumors is necessary for the following reasons: (1) Complete removal is more easily accomplished when the lesions are smaller, and (2) early removal reduces the chance of malignant change. It is hoped that in the future more such operations will be performed and reported in order to evaluate these tenets accurately. Statistics also point to a lower percentage of complete facial paralyses following total parotidectomy than that following the less radical operations of enucleation and excision.

Neoplasms

As pointed out by Lewis,[64] there are widely diversified ideas and considerable disagreement regarding prognosis and treatment of hemangiomas. He makes a plea that injection of sclerosing solutions be used, at least as a therapeutic trial, in all cases of bulky hemangioma before more radical treatment such as x-ray irradiation, radon seed implantation, or surgery. Lewis believes that the overwhelming majority of these tumors will respond satisfactorily to this conservative treatment. The more consistently good results, in his opinion, are obtained if the treatment is "hastened" in a slowly cautious manner.

For the past five years, Lewis has used a mixture of 5% sodium morrhuate with an equal volume of 2% procaine solution containing one ampule of hyaluronidase (150 units) per 5 cc. of solution. The hyaluronidase-procaine-sodium-morrhuate solution has proved very satisfactory in diffusion into the numerous blood vessels in lesions of the face. Hemangiomas involving the deeper tissues of other regions are treated just as are the single isolated tumors. Large deforming hemangiomas will require surgery in conjunction with, or following, the more conservative treatment of injection with sclerosing solution.

Kilgour[65] presents two interesting cases of hemangioma of the mandible. These are additional to eight cases recorded in the literature. In the first patient, the tumor

involved the right body of the mandible from the angle forward across the symphysis to the left canine region. The tumor was removed by excision and curettement, followed by a suitable iliac bone graft. Good occlusion and movement of the mandible resulted. In the second patient, a soft purplish mass and the exterior cortex, which was not eroded, were removed, and the cavity was packed with cancellous chips taken from the iliac crest.

Conley[66] points out that an evolving conception of composite excision and reconstruction in the treatment of cancer applies also to tumors of the head and neck. Free split-skin grafts are used to cover a wound in the head or neck area either permanently or temporarily. Free full-thickness skin grafts are valuable in many specific areas of the head and neck where definite procedures of a smaller scale and of greater refinement are indicated. Conley also discusses the use of regional pedicled flaps and of distant pedicled flaps in these regions, placement of autogenous or homologous bone into a deficient area of the mandible, and an immediate nerve graft and minor use of other grafts.

In a discussion of the surgical treatment of malignant tumors of the maxillofacial area, Stuteville[67] points out that many times a good-sized tumor of the face or neck can be excised and the defect repaired by a rotational flap. Occasionally, it is necessary to cover the defect with tissue transported from other parts of the body. The drainage nodes are removed when involved.

Briefly, Stuteville has this to say:

The maxillofacial area is the area above the clavicles, excluding the eye, the central nervous system, and the middle and inner ear.

The surgical treatment of malignant tumors of the maxillofacial area is aimed at separating the patient from his disease.

The diagnosis must be made by histopathological study.

The squamous-cell carcinomas of the oral cavity are multicentric.

There can be no set rule for the treatment of malignant tumors of this area. Each case must be considered individually.

Primary chondrosarcoma of the mandible is an extremely rare entity, as stated by Azzato.[68] In his patient, an elevated, superficially ulcerated tumor mass on the lingual side of the left alveolar ridge in the region of the second and third molars, together with the adjacent mandible, were resected. Later, the remaining upper portion of the mandible was removed with surrounding soft tissue of the infratemporal and pterygoid fossae.

Two unusual cases of nasal tumors in children are reported by Crosby.[69] A recurrent glioma over the middorsum of the nose in a child has been excised twice and a third extirpation is planned.

Both external, internal, and combined nasal gliomas occur. In the internal type, all gradations of continuity with the base of the anterior cranial fossa have been demonstrated, from complete isolation through varying degrees of fibrous and ependymal-lined stalks, to the typical encephalocele with its meningeal continuity through a cranial floor defect. The child had an external and internal glioma with broad attachment to the anterior bony septum and the under-surface of the ethmoid sinuses.

In another case of a child with a typical juvenile rhabdomyosarcoma involving the face, the entire half of the distal nose, the medial portion of the right cheek, and part of the upper lip, together with the floor of the nostril, were excised en bloc with the cautery. A sickle scalp pedicle was elevated at the same stage. The pedicle was raised, tubed, and attached to the margins of the nose defect. The forehead defect was repaired with a split-thickness skin graft from the arm. Later, a conchal cartilage graft will be implanted to maintain the normal convexity of the reconstructed ala.

Miscellanies

In presenting his philosophy of cosmetic surgery, Aufricht [70] emphasizes the justification of surgical procedures to eliminate an ugly unattractive feature. Imbalance in proportion of the nose to other facial features, folds, lines, and sagging contours create disturbing self-consciousness. Aufricht points out uncertain factors in accepting a patient for a cosmetic operation.

Snyderman [71] reports on the clinical use of bone, cartilage, dura, and fascia kept at an Auxiliary Tissue Bank at Memorial Center, New York. Supplies are furnished by the Tissue Bank of the National Naval Medical Center, at Bethesda.

Banked fascia lata grafts have been applied in closing the chest wall defect after radical mastectomy, to repair abdominal hernias, and, in one instance, to correct a seventh nerve facial paralysis. Freeze-dried bone has been used in five cases. To date freeze-dried tissue has been used in 35 cases, all of which were successful. The advantages of such tissues are that they are easily stored at room temperature; they spare the patients additional surgery necessary for removal of the grafts, and the graft, being an organic material, can be assimilated or replaced by the body.

Prudden, Nishihara and Baker [72] investigated the local effect of cartilage on wound healing by testing the wound tensile strength in albino rats. Paired animals for testing and control which exhibited wound infection were rejected. The effect of bovine tracheal cartilage, bone flour, talcum powder, gelatin, and parenteral methione upon the tensile strength of the wounds was tested on the seventh postoperative day. A modified technique of Meadows and Prudden was used. A rotary pump was connected with a latex condom, which was inserted into the peritoneal cavity of the killed animals through a defect in the apex of the vagina. The pressure at which the wound split and the air-filled condom extruded itself through the defect was recorded. Only cartilage powder produced significant and unique increase in tensile strength of wounds under normal nutritional and endocrine circumstances. The specific fraction of cartilage responsible for this effect is unknown, being as yet unseparated and uncharacterized.

120 Halsted St.

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Abstracts from Current Literature

Ear

TREATMENT OF IMPACTED CERUMEN: CLINICAL EFFECTIVENESS OF A NEW CERUMENOLYTIC. MEYER BERGER and HARRY S. BIKOFF, New York J. M. 59:1794 (May 1) 1959.

Ceruminal impaction occurs in 18% of all adult males and "can have far-reaching and serious sequelae." Factors controlling "cerumenogenesis" and "cerumenolysis" are not completely known, but anatomic abnormalities of the canal, areas of inflammation in the surface epithelium, and disproportions in the composition of the elements composing cerumen are stressed. "It is possible for the retained cerumen to become impacted and lead to *keratosis obturans* . . . removal of this mass . . . is often a difficult procedure. This study selected 47 children and 23 adults." The new agent (name not mentioned here) is highly praised. It simply dissolves the wax so that it can be readily washed out with plain water. The authors speak of "the inordinate length of time . . . thirty minutes on one ear alone" consumed by other methods, and of "severe pain" caused by removal of the wax, which may also be sufficient to rupture the tympanum. "Bleeding is common, and there is usually some dermatitis from impacted wax. . . . Removal of cerumen by the accepted methods is a time-consuming and generally ineffective process. . . . From this study we would conclude an unusually high percentage of efficacy, the ease of administration, the total lack of adverse reactions, warrant its use in all cases of excessive and impacted ear wax."

ABSTRACTOR'S COMMENT: Why this sudden interest in ear wax? This is the second article I have seen in two different journals since March, 1959, and, *mirabile dictu*, each has been promptly followed by "literature" from companies which have the product for sale to physicians! Strange coincidence or—? Each "study" aims to be thoroughly scientific. Everything is examined but blood and urine. Perhaps this should also be done in every case of "ceruminogenesis." It would at least be part of a "complete physical." In 50 years of otology, I have never met with complications in removal of wax save the very efficient efforts of a crying, obstreperous child to keep the examiner from seeing or doing anything. Even a "mummy wrapping" cannot hold the head still. In an occasional patient I have used primary anesthesia, but this is uncommon. As for rupture of the tympanum through unskillful handling, this must be a *rara avis*—I have never seen or heard of it till now. What I have seen, very recently, was erysipelas following instrumentation with injury to the canal wall. This last borders very closely on malpractice. Too often, removal of wax is left to a nonmedical attendant. Even so, she or he can be trained to look in at intervals with an electric otoscope to make sure that removal is complete. In any case, no patient should leave the office without being reexamined by the doctor. For years I have used a saturated solution of sodium bicarbonate to be instilled at home, often for 24 hours or more. Then a return to the office has brought forth the wax very readily. A rubber 4 oz. hand syringe, fitted with an eye dropper (bulbous end), is quite efficient. I have never thought it necessary to make laboratory studies of the cerumen.

I. W. VOORHEES, New York.

USE OF POLYETHYLENE PROSTHESES IN MOBILIZATION OF STAPES SURGERY. ALAN AUSTIN SCHEER, New York J. M. 59:2169 (June 1) 1959.

Scheer works in the Manhattan Eye, Ear, and Throat Hospital. He has developed a procedure for removal of the head and crura of the stapes, replacing them by a tube of polyethylene which makes contact with the incus and the stapes—a sort of "go-between." Mobilization of the stapes is much in vogue just now but is often beset by incidental fracture of the crura. "It has been difficult to salvage hearing for those patients who have had crural fractures." Scheer's first step after cutting the stapedius tendon is to remove the broken crura and head of the stapes. The footplate must then be mobilized. Proper length of the prosthetic prop is difficult to judge. It may be either too long or too short. If so, it will not accomplish the hoped-for results. Assuming that the length is correct, one end is attached to the free end of the incus; the other end leans against the footplate. Care must be taken not to force the footplate off balance, that is, to have it press against the labyrinthine mem-

brane improperly. Any complications will be due to trauma to this membranous labyrinth. Some 80 patients have been operated upon with a good degree of success in restoration of hearing. Contraindications are as follows: if the footplate is in multiple segments as a result of prying it free or if the annular ligament at the superior or inferior margin of the footplate tears so that an uneven edge invades the vestibule "like an open door."

I. W. VOORHEES, New York.

THE BLUE TYMPANIC MEMBRANE. J. J. DEBAIN, Ann. oto-rhino-laryng. Paris, p. 57, 1958.

The author discusses a rather unusual condition, namely the blue tympanic membrane. This may be due to alterations of the tympanic membrane per se. This may occur in grippal conditions, dilatations of the veins of the tympanic membrane, angiomas of the membrane, and interstitial hematomas. They also occur in congenital cardiopathies. A second group is where there is a marked prominence of jugular bulb which extends high into the tympanic cavity, and owing to its presence either with or without a dehiscence of the bone covering the jugular bulb, the appearance of this bluish color through the tympanic membrane is apparent. A third group consists of new growths within the tympanic cavity which may be either a glomus type of a tumor (it may be angiomas or similar conditions). Fourthly, there is hemotympanum, which may be due to a vascular accident, lead poisoning, various types of cardiopathies, malignant tumors of the tympanic cavity, packing of the nasal cavity for epistaxis, and hemolytic purpura. There may also be a hemotympanum due to aerotitis, to fractures of the petrous tip, and a number of cases that the author labels idiopathic hemotympanum.

The treatment of these conditions is first a paracentesis of the tympanic membrane to determine whether or not this is a simple pathology that involves either the tympanic membrane or the tympanic cavity, or whether there is some pathology within the middle ear cavity. If there is a suggestion that there may be a new growth, then surgery is indicated. An atticoantrotomy is performed in one or two stages. Radiotherapy is of no value. The prognosis depends upon the underlying pathology.

In conclusion, the author pleads that if this condition comes to the attention of the otologist it may be well for him to report the case, since his own bibliography is extremely limited—to 20 cases.

PERSKY, Philadelphia.

NEW OBSERVATIONS CONCERNING LABYRINTHINE VERTIGO TREATED BY L-ACETYL-DL-LEUCINATE ETHANOLAIMINE. G. DUCHESNAY and P. PIALOUX, J. franç. oto-rhino-laryng. 7:991 (Nov.-Dec.) 1958.

The authors have evaluated 33 cases of vertigo (14 of which were true Ménière's disease), 5 cases of post-traumatic vertigo, 7 cases of postural vertigo, and 5 cases who presented vertigenous symptoms of toxic origin, usually after streptomycin therapy. These patients were treated by a preparation of *l*-acetyl-DL-leucinate ethanolaimine, 33 of which received intramuscular injections of 500 mg. a day, and in 3 cases, received either 1 gm. or 1.5 gm. a day by either the buccal or rectal route. They have obtained improvement and remissions in 25 of these patients. The 14 cases with true Ménière's disease showed that 12 were completely improved. Four of the five cases of post-traumatic vertigo showed improvement; however, in the poststreptomycin group they had three complete failures and two improvements. The principle action of this medication is primarily a sedative. Its greatest results have been shown to be in the true Ménière's complex.

The authors feel that this new medication has a tremendous amount of efficacy and should be employed where the usual routine treatments such as hypotonic solutions, the antihistamines, the sympatholytics, Heperine, vitamin B therapy and Novocaine have not been successful. In the few cases where there was apparent failure of a complete cure, the authors believe that the dose was either insufficient or the treatment was limited. They recommend this new treatment both as an adjunct to other measures or as an independent method of treatment, which can produce excellent results in the control of these symptoms.

PERSKY, Philadelphia.

Larynx

BRONCHOGENIC CARCINOMA SIMULATING THYROIDITIS. MORRIS KLATZKO, New York J. M. 59:2423 (June 15) 1959.

Klatzko states that bronchogenic carcinoma ranks with prostatic and stomach lesions among the commonest cancers found in the human male, but it may be mistaken for symptoms seen in

brain tumor, stroke, bone tumor, or even Addison's disease! A 62-year-old white man came in with painful swelling of the neck, dyspnea, cough, hoarseness, and a weight loss of 10 lb. Examination of the thyroid gave every indication of subacute thyroiditis. History of carcinoma in the family was evidential. The man had been "gassed" in World War I, had been alcoholic 10 years previously but was then free, and smoked a pack of cigarettes a day. The most striking item was a hard supraclavicular node which on section proved to be carcinomatous. This gave the first hint that it might be metastatic from lung involvement, but this is so unusual that it was merely possible but not probable. In spite of thyroid studies and treatment (prednisolone, etc.) the patient went on to death. Autopsy showed that the thyroid was fairly replaced by carcinomatous tissue. There was a small bronchogenic area in the left upper lobe of the lung. From this had come the metastasis to the thyroid and supraclavicular left fossa. Mediastinal nodes were, of course, present, but recognized only at necropsy.

ABSTRACTOR'S COMMENT: This is a careful and complete study and report of an unusual case of primary lung cancer with metastases in the neck. It illustrates how misleading diagnosis can be.

I. W. VOORHEES, New York.

Pharynx

PHARYNGEAL PARESTHESIAS AND GLOSSODYNIA. R. MADURO, Ann. oto-rhino-laryng. Paris, p. 141, 1958.

These troublesome conditions appear more frequently in private practice than in hospital consultation. They are characterized in a number of ways. One is a painful sensation either of the tongue situated rather superficially; deeper, where one suspects an involvement of the superior laryngeal nerve; to the lateral side of the oral pharynx; on either one side or the other in the region of the tonsils, or on the soft palate. The second sensation is that wherein the patient complains of a sensation of a inspissated plug of mucus on the posterior surface of the pharynx. The third group are those that complain of a pharyngeal tenesmus. The fourth group complained of a sensation of a mass in the oropharynx. Lastly, there is the sensation of foreign bodies in the pharynx. The author classified these symptoms in two groups: (1) those that are purely psychogenic and (2) those that are due to organic factors either in the mouth or the pharynx.

From the organic standpoint, there may be some type of pathology either in the sinuses, the oropharynx, or the pharynx. There may be hypertrophy of the serous or mucous glands. There may be an atrophy of a great many of these glands. There may also be some general pathology in the sinuses which is responsible for these symptoms. The treatment should be directed to the sinuses and the posterior pharynx. Those patients complaining of having a sensation of a "ball" in their throat may present either a very small cyst or a small adenoma of the thyroid, which extends into the pharynx. Attention to this should be considered. Pain on either lateral side of the pharynx in the vicinity of the tonsils may be due to an involvement of the styloid process. Pharyngeal tenesmus and neuralgia may be due to some irritation of the superior laryngeal nerve. Careful evaluation of the larynx is important. There may be a number of causes, such as dental origin, or an arthrosis of the cervical spine. There may be syndromes that involve either the upper portion, the middle portion, or the inferior portion of the pharynx. Occasionally it may be necessary to anesthetize the trunk of the superior laryngeal nerve with Novocaine to reduce or eliminate these sensations. Another group of glossodynias are what the author terms superficial sensations. They may be occasioned by the ingestion of acid foods and spices and the absorption of various alcoholic beverages. They may also be due to a chemical change produced by a prosthesis or a dental process, such as the metallic inlay.

Another group of glossodynias can be found in certain types of anemias, particularily in hyperchromic pernicious anemia. This has been designated as the glossitis of Hunter. It may also be found in some of the essential hypochromic anemia, and, lastly, there is a group of glossodynias that are associated with vitamin B or riboflavin deficiency.

The author has tried to indicate a few of the problems that must be investigated in attempting to determine some of the etiologic factory that may be responsible for these frequent and distressing symptoms.

PERSKY, Philadelphia.

Miscellaneous

GROWTH SUPPRESSION IN ASTHMATIC CHILDREN RECEIVING PROLONGED THERAPY WITH PREDNISONE AND METHYLPREDNISOLONE. THOMAS E. VAN METRE JR. and HERMAN L. PINKERTON JR., J. Allergy, Vol. 30, No. 2 (March-April) 1959.

A study was made on 13 children with intractable perennial asthma to determine whether adrenal cortical hormones of the cortisone type can suppress linear growth in children. Twelve of the thirteen showed linear growth retardation roughly proportional to the average dose employed. Normal or accelerated growth rate appeared when therapy was discontinued or the dosage reduced. A similar study conducted on 52 children with perennial bronchial asthma who received no corticosteroid therapy showed linear growth to be essentially normal, indicating that chronic bronchial asthma of itself does not cause growth suppression.

Clinicians are cautioned to observe the growth of children receiving prolonged steroid therapy. The dosage should be reduced below growth-suppressing levels when necessary to assure recovery of the growth pattern to the normal level.

MISSAL, Cleveland.

SOME PRESENT CONCEPTS OF CARCINOGENESIS. C. P. RHOADS, W. E. O'DONNELL, J. J. NICKSON, C. M. SOUTHAM, and W. E. WHITMORE, New York J. Med. 59:2154 (June 1) 1959.

This symposium, with Cornelius P. Rhoads of Sloan-Kettering Foundation as Moderator, was presented at the Annual Meeting of the New York State Medical Society, in May, 1958. It was a general overlook of the entire cancer problem. The part of most interest to ENT men is that which has to do with inhalants, late radiation injury, and the role of viruses. All of the participants are connected with Cornell.

At this time every person living is exposed to the influence of inhalants, especially with regard to cancer. Miners in Europe have been studied in this relationship. They are exposed to radioactive dust, but how do the inhalants act? Such substances as arsenic, chromate, asbestos, nickel, certain petroleum products and beryllium are set down as likely to be carcinogenic and may play a role in one-quarter of all cancer deaths. Air pollution is due to incomplete combustion of solid, liquid, or gaseous fuels. They must be studied from standpoints of epidemiology, chemistry, and human tissues. Urban populations are, of course, more exposed to such influences than rural ones. A well-conducted epidemiological or statistical study is "every bit as valid as the so-called scientific experiments of laboratory evidence." Two methods are in use: the retrospective and the prospective. The former is historical in the sense of what has happened in the past to an "average population." Over 20 studies have been made in eight different countries with respect to lung, oral, and laryngeal cancer. Strikingly similar conclusions have been reached in all of these. They show clearly that smoking tobacco is an important factor in the genesis of cancer as compared with nonsmoking. Lung cancer, for example, of the epidermoid variety is rare among nonsmokers.

The prospective method works differently, since it concerns what may happen in a given group. The American Cancer Society in 1951 identified a group of over 200,000 white American men between 50 and 69 years for 44 months as to causes of death. Cigarette smoking was studied as to amounts (packs a day). Mortality regarding lung cancer among the two-packs-a-day smokers was 62 times that of nonsmokers regardless of city or urban residence! Air pollution has to be considered also but is extremely difficult to differentiate.

Animal studies have to do with the amount and effect of tar in carcinogenesis. The skin of mice is vulnerable to tumor formation, both benign and malign when exposed to tar applications.

Chemical studies show that the hydrocarbons are notably carcinogenic, the aromatic hydrocarbons especially, but here cause and effect have not been too well established to date.

Human tissue studies have been more informative. Auerbach and his associates have studied specimens from the tracheobronchial tree from patients with or without lung cancer. It was determined that one could follow the stages from normal epithelium to actual carcinoma in situ—in some cases actual invasive carcinoma. In summary, tobacco is a carcinogenic agent; "in its absence at least 50 or perhaps 80 percent of ca in the respiratory tract would not occur." The thought is to remove all irritating matter from tobacco so that carcinoma incidence is reduced to a minimum.

Dr. Nickson spoke on late radiation injury, particularly as to "fall-out." In 1902 it was found that carcinoma of the skin was frequent among workers with radiation exposure (54 cases had been reported). Osteogenic sarcoma also came to light in 1925 in the famous case

of watch-dial painters. The induction time was slow, from 15 to 20 years, but was irreversible. As for "cancer of the blood," leukemia, Dr. Nickson says, "I think there is no doubt that leukemia can occur as a result of occupational exposure to radiation." The condition has developed from therapeutic use of ionizing radiation.

The role of viruses was described by Dr. Southam. There are tumors found in rabbits, mice, and fowl evidently caused by viruses, and yet we are not ready to assume the virus origin of cancer in general. A virus is a submicroscopic, obligate, intracellular parasite which intrenches itself in the cell body and is, therefore, relatively free from extracellular antibodies or chemical agents which could destroy it. Moreover, it may go right along with cell division and thus be self-perpetuating. Koch's postulates are against the theory of viruses as a cause of cancer, which is not one disease but "a tremendously large group of diseases," and so a single causative factor is not presently to be considered.

Unfortunately, in spite of all the work that has been and is being done to unveil the cancer mystery, we must admit that it is still an unsolved problem. However, the outlook is not hopeless; some day we shall know the cause or causes of all cancers and be able to reduce its influence as the horrible killer it now is.

I. W. VOORHEES, New York.

AGRANULOCYTOSIS FOLLOWING ANTIHISTAMINE THERAPY: REPORT OF A FATAL CASE. BENJAMIN F. NORRIS, New York J. Med. 59:2600 (July 1) 1959.

Norris reports this case from St. Peter's Hospital, Albany, N. Y., and the Department of Hematology, Bender Hygienic Laboratory.

A 64-year-old woman appeared with a pruritic rash, but her distressing symptoms were abdominal pain, nausea, and diarrhea. The throat showed a "hemorrhagic area about 1 cm. in diameter on the left soft palate." Throat culture yielded Hemophilus influenzae, Staphylococcus pyogenes var. alba, and Neisseria catarrhalis. Blood culture was negative. Although there was a presumption of allergy, it is not so stated, but diagnosis on admission was "a possible strangulated left inguinal hernia." A prescription was given for Parabromdylamine Maleate. She seems to have taken this from about the middle of May to Feb. 21, apparently without a prescription or permission for renewal. Without relief, she consulted another physician. This adviser prescribed Thenalidine Tartrate, a 25 mg. tablet four times a day—24 in all. Again, she seems to have renewed the quantity without medical advice; then she died. Autopsy findings helped little to establish a firm diagnosis. Nothing is said of the left lower quadrant condition of "strangulated hernia." Blood culture post mortem showed "a good growth in pure culture of Escherichia coli." This did not appear in culture No. I, or is not so mentioned. . .

ABSTRACTER'S COMMENT: Occasionally a throat specialist can make a tentative diagnosis of agranulocytosis from inspection, plus an immediate blood culture, but thorough laboratory work later on disclosed this condition without showing cause-and-effect in relation to the throat . . . Those working in allergy need to be especially cautious of the powerful newer remedies when long continued. In this case self-treatment seems to have been contributory to death.

I. W. VOORHEES, New York.

Correction

In the article by F. Bertram Briess entitled "Voice Therapy" which appeared on pages 61-69 of the January, 1959, issue of the ARCHIVES, the legend for Figure 1 should read as follows:

Fig. 1.—In the health phonation, the lowest tone of the natural voice compass may consist of 90% thyroarytenoid and 10% cricothyroid function, while the highest tone may be compounded of 10% thyroarytenoid and 90% cricothyroid function.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. G. Arnold Henry, 170 St. George St., Toronto.
Secretary: Dr. Donald M. MacRae, 324 Spring Garden Rd., Halifax, N. S.

Venezuela Otorhinolaryngology Society

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General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

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Place: Groningen, Netherlands.

Mexican Society of Otolaryngology

President: Dr. Rafael Giorgana
Secretary: Dr. Carlos Valenzuela, Monterrey 47-201, Mexico 7, D. F.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Paul H. Holinger, Chicago.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Seventh Congress, Americana Hotel, Miami Beach, Fla., March 20-23, 1960.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Paul H. Holinger, Chicago.
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President: Dr. John H. Dunnington, 635 W. 165th St., New York.
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* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3.
Meeting: Deauville Hotel, Miami Beach Fla., March 15-16, 1960. Lake Placid Club, Essex County, N.Y., May 20, 1961. Sheraton-Dallas Hotel, Dallas, Texas, May 1-2, 1962. Boca Raton Hotel, Boca Raton, Fla., April 2-3, 1963.

American Laryngological Association

President: Dr. William J. McNally, Montreal.
Secretary: Dr. Lyman G. Richards, Wellesley Hills, Mass.
Meetings: Deauville Hotel, Miami Beach, Fla., March 18-19, 1960. Lake Placid Club, Essex County, N.Y., May 21-22, 1961. Sheraton-Dallas Hotel, Dallas, Texas, May 4-5, 1962. Boca Raton Hotel, Boca Raton, Fla., March 31-April 1, 1963.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Theodore E. Walsh, 640 So. Kingshighway, St. Louis, Mo.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester 7, N.Y.
Meeting: Deauville Hotel, Miami Beach, Fla., March 15-17, 1960. Lake Placid Club, Essex County, N.Y., May 23-25, 1961. Sheraton-Dallas Hotel, Dallas, Texas, May 1-3, 1962. Boca Raton Hotel, Boca Raton, Fla., April 2-4, 1963.

SECTIONS:

Eastern.—Vice President: Dr. F. Johnson Putney, Philadelphia, Pa.
Meeting: Warwick Hotel, Philadelphia, Pa., Jan. 8, 1960.
(Council—Biltmore Hotel, Jan. 9, 1960).
Southern.—Vice President: Dr. Edley H. Jones, Vicksburg, Miss.
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1. Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics, ed. 2, New York, The Macmillan Company, 1955, p. 69.

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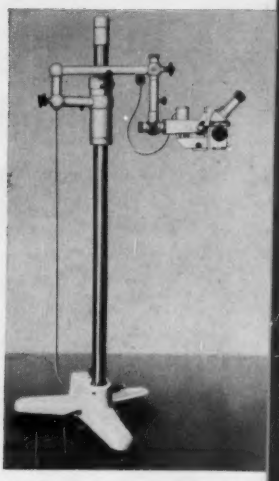

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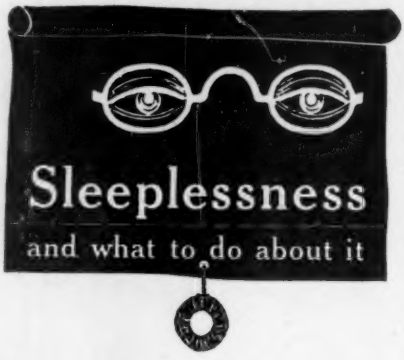

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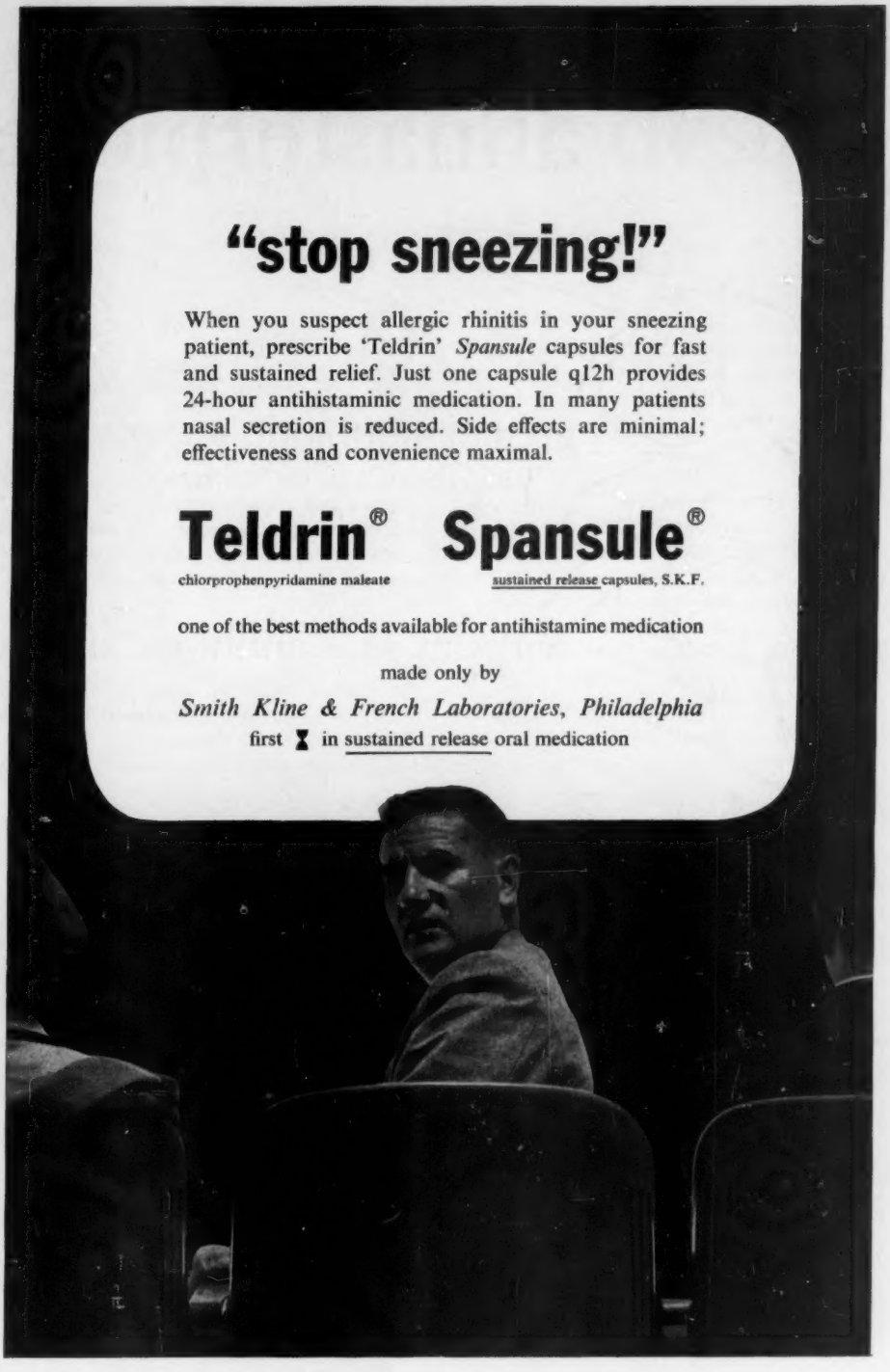
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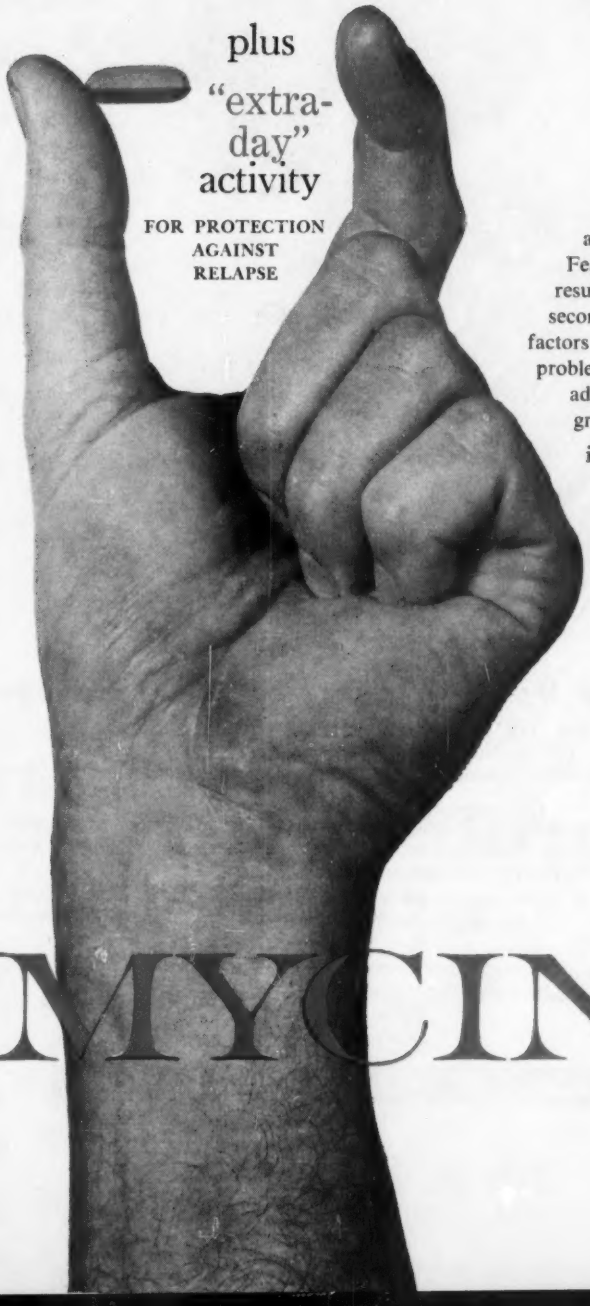

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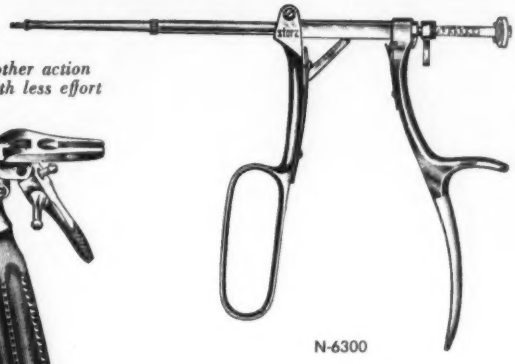

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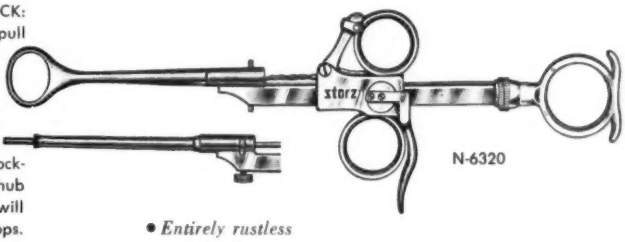

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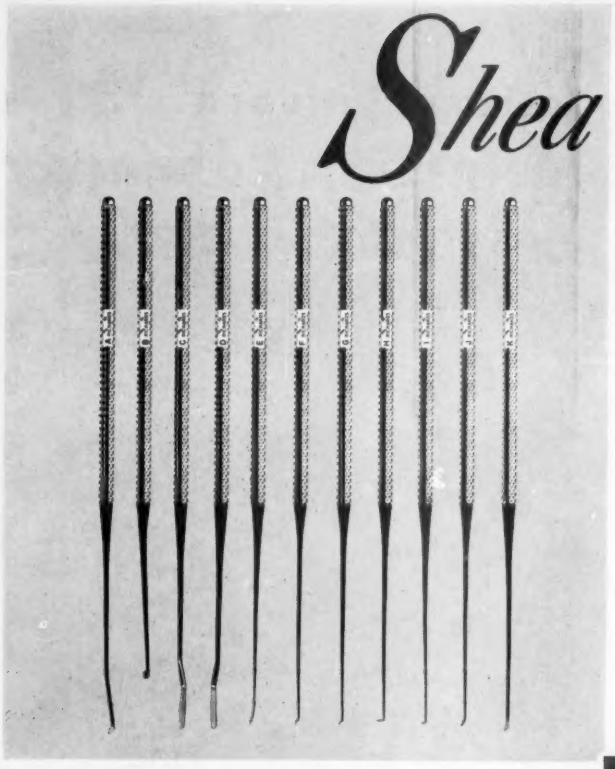

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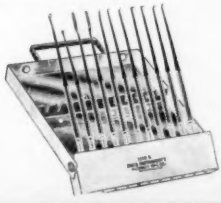

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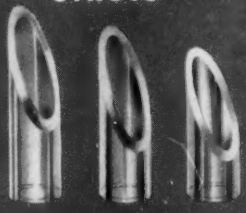

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